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THE EARLY DIAGNOSIS OF DISEASES OF THE CHEST*

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THE early diagnosis of diseases of the chest is a matter of more importance to the physician today than at any time in the history of medicine. This is true for the following reasons. Intrathoracic abnormalities and diseases are more frequent than has ever been suspected before, as has been revealed by mass x-ray surveys in recent years. Modern diagnostic procedures make early diagnosis more easily attainable than ever before. Modern medical and surgical procedures offer a better chance of control or cure in most diseases of the chest than ever before. Early diagnosis, which is essential to successful therapy, is being made in the minority rather than in the majority of patients with intrathoracic disease. Lastly, evidence points to the failure on the part of physicians and hospitals to utilize modern methods of diagnosis as the outstanding cause of this unfortunate state of affairs.

Not so long ago the physician had to rely on the history, the physical examination and inadequate bacteriologic study of the sputum to establish a diagnosis of intrathoracic disease. As is well known, diagnoses were made during this era in the vast majority of cases only when the pathologic process was in a moderately advanced or far advanced stage. In addition, the available methods of treatment were extremely limited and of questionable effectiveness. These facts led to the hopeless attitude of the medical profession and laymen alike toward these maladies. In recent years, however, great strides have been made in the diagnosis and therapy of diseases of the chest. Fluoroscopy and roentgenography have become universally available. More recently, mass surveys with miniature film taken with photofluorographic technic have brought onto the horizon the possibility of x-raying the chest of almost every man, woman and child in a given community at a cost of only a few cents per person. Bronchoscopy has been perfected so that it can now be performed with only slight discomfort to the patient and with little risk. Better bacteriologic technics, including the examination of pooled specimens of sputum and fasting gastric contents for tubercle bacilli, have been developed. Aspiration biopsy of lung tumors is a helpful procedure in certain well-selected cases. The advance in medical and surgical therapy of chest lesions has kept pace with the diagnostic developments and provides control of the disease process or the cure of many patients, provided that the diagnosis has been made early.

In spite of the fact that the tools with which to work are now available, there is overwhelming evidence from mass surveys and the analysis of case histories that the early diagnosis of intrathoracic disease, as already stated, is accomplished in the minority rather than in the majority of cases. Sixty per cent of the tuberculous patients in this country who are referred to sanatoriums have far advanced disease, and only 10 per cent have minimal lesions. Less than 25 per cent of the patients with cancer of the lung are referred for surgery before metastases or extension of the tumor has occurred. In a study of 153 patients with cancer of the lung, Overholt[1] discovered that in 95 cases (60 per cent), not only had an incorrect diagnosis been made, but also treatment based on it had been maintained for long periods of time. The average patient went to the doctor three months after the first symptom occurred. It required an average of three and a fourth months for the physician to request the first x-ray film of the chest. The correct diagnosis was not established until another six months had passed. Thus, definitive therapy was delayed an average of nine months from the time of the first visit and — what is more alarming — for six months after the roentgenogram had revealed the lesion. In other words, only three months of the delay was due to the patient and nine months was due to the failure of the physician to establish the proper diagnosis early.

A study of this problem has suggested several reasons for the delay in diagnosis. The patient frequently delays going to the physician because the incipient stage of the disease process is so frequently

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associated with few or no symptoms; the presenting clinical picture often suggests another diagnosis; physical examination is notoriously unreliable because of the paucity or absence of physical signs; and the application of rigid diagnostic methods is often delayed because the physician has not developed a sufficiently strong suspicion of the underlying disease.

Misleading Nature of Clinical Picture

The early clinical course of intrathoracic lesions is apt to be misleading because symptoms are either absent or extremely mild or masquerade as some other disease process. The important clinical fact that physicians must come to recognize more fully is that there is almost always an early asymptomatic stage, during which the process can be discovered only by routine roentgenogram of the chest. When symptoms do occur, they usually point to disease in the chest, but they are apt to be so mild that the life-threatening cause is not suspected. As a result, many of these patients are treated for bronchitis, the common cold, grippe and so forth.

The masquerading character of carcinoma of the lung has been emphasized by Alexander[2] and Overholt.[1] Just how important a factor this is can be gleaned from the fact that 60 per cent of 153 patients studied by Overholt were treated on the basis of an incorrect diagnosis. These diagnoses included tuberculosis, unresolved pneumonia, lung abscess, bronchitis, asthma, heart disease, pleurisy, metastatic cancer and arthritis. Benign lung tumor may also present a similar bizarre clinical picture.

The masquerading character of pulmonary tuberculosis has not been so well described in the literature, but during the last few years I have seen tuberculosis simulate the clinical picture of bronchiectasis, abscess, asthma, pneumonia and carcinoma, as well as other less well defined clinical pictures, such as bronchitis and virus pneumonia. Recent bronchoscopic studies have revealed that 10 per cent of patients with pulmonary tuberculosis also have involvement of the major bronchi.[3] As a result, stenosis is not infrequent and, like cancer, may cause all degrees of bronchial occlusion, with resulting obstructive emphysema, atelectasis and suppuration. Wheezing is a usual complaint of these patients, and they often seek advice because they believe that they have asthma. The wheezing, however, is always unilateral, and this should serve to differentiate it from true asthma.

Pleurisy, with or without effusion, spontaneous pneumothorax, persistent hoarseness or anal fistula may be the first condition of which the patient with pulmonary tuberculosis complains. All such patients should have chest roentgenograms to rule out pulmonary tuberculosis. In those with pleural effusion, the pleural fluid should be studied for tubercle bacilli by examination of a concentrated specimen, culture and guinea-pig inoculation. Because of the serious prognostic significance of pleural effusion, especially in young people, these patients should be treated as if they had parenchymal tuberculous lesions and followed routinely by frequent x-ray films of the chest. As Kallner[4] found, 39 per cent of patients with pleural effusion who were denied sanatorium therapy developed pulmonary tuberculosis, usually within five years, and 22 per cent of these patients died. This is in striking contrast to the good prognosis of such patients when properly treated, as reported by Trudeau.[5]

No attempt will be made to cover completely the symptoms associated with intrathoracic diseases, but the following points require emphasis. Almost all the diseases of the chest have an early asymptomatic stage, during which the pathologic process can be discovered only by x-raying the chest. This stage is apt to be so mild that the seriousness of the underlying lesion is overlooked. The masquerading tendency of intrathoracic disease, especially cancer and tuberculosis, has been emphasized. They may masquerade as each other, or as any of the commonplace diseases of the chest, or as an entirely foreign clinical picture, such as arthritis.

Unreliability of Physical Examination

Physical examination of the chest is an essential procedure in the evaluation of any patient, especially those with diseases of the chest. Relinquishment of such procedures is not advocated, but it must be emphasized that the time has come to recognize their limitations as well as their value. As early as 1933, Sampson and Brown[6] reported that the staff of the Trudeau Sanatorium had concluded that moderately coarse rales at an apex were the only reliable data obtained on physical examination, and added that these were present in only 27 per cent of the minimal cases. They analyzed the occurrence of the five cardinal signs and symptoms of tuberculosis in a series of 280 cases with minimal disease. Tubercle bacilli were found in the sputum in 35 per cent, rales in 27 per cent, hemoptysis in 26 per cent, pleural effusion in 12 per cent, and x-ray evidence of pulmonary tuberculosis in more than 99 per cent. These pioneers pointed the way to the early diagnosis of pulmonary tuberculosis over a decade ago, yet in the everyday practice of the country and in the teaching of medical students methods of diagnosis are predominantly employed that cannot claim more than a 25 to 30 per cent efficiency. This partially explains why 85 to 90 per cent of patients with pulmonary tuberculosis are diagnosed only when they have reached the moderately advanced or far advanced stages.

In respect to carcinoma of the lung, physical signs are usually absent in the very early stages. Unilateral wheeze and suppression of breath sounds due to obstructive emphysema are the most frequent early signs. When bronchial obstruction is more complete, signs of atelectasis, consolidation or ab-

scess may be present. It is only fair to state that physical signs are extremely unreliable and at best only suggestive in cases of pulmonary cancer.

Value of Routine Fluoroscopic Examinations

In 1927, the Metropolitan Life Insurance Company made fluoroscopic examination of the chest a routine supplement to each physical examination at its home office.[7, 8] This enabled the examiners to discover intrathoracic abnormalities and disease that would have been missed by physical examination and history. As a result, treatment was started in the majority of cases before physical signs or symptoms had become apparent.

Block and Tucker[9] recently reported to the American Trudeau Society their experiences with routine fluoroscopic examination of the chest of all patients admitted to the University of Chicago Clinic. The results of this admirable clinical study, which was applied to 15,000 cases, were remarkable. Positive findings were revealed in 3187 cases (21.3 per cent). The incidence of tuberculosis in this group of patients was 4.2 per cent, that of clinically significant tuberculous lesions 1.4 per cent, that of tumors of the lung 0.6 per cent, that of mediastinal and diaphragmatic disease 0.6 per cent, and that of cardiovascular abnormalities 14.4 per cent. In addition, there were other significant findings, such as pleural disease, anomalies, skeletal disease, foreign bodies and substernal thyroid glands. The significant fact concerning the pulmonary tuberculosis discovered by this method was that two thirds of the patients had minimal lesions, one sixth moderately advanced, and one sixth far advanced lesions. This represents a reversal of the distribution of the stages of tuberculosis in patients referred from private practice to sanatoriums. On the basis of this study Block and Tucker estimate that in the fifteen years prior to the institution of routine chest fluoroscopic examination, 3000 cases of active tuberculosis were missed in their clinic in spite of careful history-taking and physical examination. They also state that in the coming year about 600,000 patients with pulmonary tuberculosis will undergo physical examination in hospitals and physicians' offices of this country without a diagnosis being established.

These data demonstrate the value of routine fluoroscopic examination of the chest as a supplement to the physical examination. They further show the inefficiency and inadequacy of the history and physical examination in apprehending intrathoracic abnormalities in the early stage of development.

Value of Routine Roentgenograms

Stereoscopic 14-by-17-inch films remain the most ideal means of demonstrating lesions of the chest, but the prohibitive cost of this procedure, together with the demand for a cheaper and less cumbersome form of chest roentgenography for mass surveys, has led to the development of miniature films taken by photofluorographic technic. The 35-mm. and 4-by-5-inch films are the ones in general use. Plain or stereoscopic views may be taken. Birkelo[8] has demonstrated that the 4-by-5-inch film can be used in mass survey work with only a 2 per cent margin of error, as compared with results obtained with the regular 14-by-17-inch film.

The utilization of this technic in mass surveys has served to demonstrate that intrathoracic abnormalities, neoplasms and infections are much more frequent than has been assumed from available clinical or autopsy data. Under the direction of Hodges,[10] of the University of Michigan Hospital, every patient admitted to the clinic since July 1, 1941, has had the benefit of a 35-mm. photofluorographic examination. Thorough roentgenologic study has been recommended for any patient whose miniature film reveals a significant abnormality. During the first four months of this work, 7841 patients were examined. Of these, 74.0 per cent had no abnormality, 14.1 per cent had deviations from normal not considered to be clinically significant, and 9.3 per cent had abnormalities demanding more extensive roentgenologic study. Hodges states, "Photofluorography employed to survey the chests of all patients regularly registered in hospitals and clinics can be expected to disclose signs of significant thoracic disease in 8 to 10 per cent of the patient group."

The foregoing data demonstrate conclusively that some form of roentgenologic study of the chest should be a routine procedure in the examination of every patient admitted to hospitals and institutions. Routine blood-cell counts reveal blood dyscrasias in a small fraction of 1 per cent of cases, routine urinalysis reveals diabetes in about 0.4 per cent, and routine serologic tests reveal syphilis in about 2 per cent. These routine laboratory procedures have been required for years in every good clinic and hospital. Yet, today, these same institutions are neglecting a procedure that will uncover 8 to 10 per cent significant thoracic disease. In the light of present knowledge, routine roentgenologic study of the chest is at least four times as efficient and important as any other routine procedure now in use. In addition to its value as a diagnostic aid, it serves to protect patients and the hospital personnel against the unsuspected active cases of tuberculosis that are constantly present in hospitals without any isolation technic whatsoever being applied. When either fluoroscopy or photofluorography is used, the procedure requires little time and can be done at a cost of only a few cents a patient. The additional expense can be defrayed by a minimal charge added to the laboratory fee.

Value of Sputum Studies for Tubercle Bacilli

In every patient suspected of having tuberculosis careful sputum studies should be performed in an

attempt to confirm the diagnosis. A positive sputum, however, is not an absolute requirement for the diagnosis of pulmonary tuberculosis. A positive sputum may never be found in some patients with minimal lesions and in others with inactive disease.

Failure to secure positive results from sputum examination is often due to the fact that the technics applied are not sufficiently rigid. There are two important factors that determine the efficacy of sputum studies — the length of time over which the sputum specimen is collected and the method of examination. The routine smear for tubercle bacilli is notoriously unreliable. Twenty to 30 per cent of the sputums negative on direct smear are found to be positive when the sputum is examined by the concentration method. Pooled specimens, with the sputum collected over a period of three to seven days, further increases the positive findings. Multiple cultures and guinea-pig inoculation of the sputum and gastric contents should always be employed before one decides that the sputum is negative.

To avoid delay in diagnosis, the following routine is suggested. First, the use of routine smears should be avoided. These smears are so unreliable that a negative result is meaningless. Second, three consecutive seventy-two-hour pooled sputum specimens should be examined by the concentration method. If they are negative on microscopic examination, the sediment should be planted on a culture medium and inoculated into guinea pigs. Third, if the seventy-two-hour specimens are negative on microscopic examination, the next step is the immediate examination of three consecutive gastric lavages by the concentration method. Acid-fast bacilli are often found in gastric lavages when the sputum is negative, especially in children and young women. The gastric specimens should always be cultured and inoculated into a guinea pig to identify the acid-fast bacilli as tubercle bacilli.

Utilizing all available methods of examination, Pinner and Werner[11] have found that over 99 per cent of patients with active tuberculosis have tubercle bacilli in the sputum. Thus, when these tests are repeatedly negative in a patient with a demonstrable parenchymal infiltration in the lung that is apparently active, the lesion is probably nontuberculous, and other diagnostic procedures are indicated.

Diagnostic Procedures in Patients with Suspected Cancer of Lung

Any patient with a visible tumor or an unexplained density or suppuration in the lung, especially if he is in the middle or older age group, should be suspected of having a pulmonary cancer. When such lesions are discovered in the minimal, asymptomatic stage, establishment of the diagnosis is most urgent, because at this stage metastases and extension of the tumor are less likely to have occurred and surgical extirpation offers a greater chance of cure. Such patients should be bronchoscoped immediately; 60 to 70 per cent of bronchiogenic carcinomas originate in the major bronchi, and a biopsy specimen to establish the diagnosis can be obtained. All patients in whom the biopsy is positive should be explored at once, provided that no clinical evidence of metastasis or extension of the tumor is present.

If the bronchoscopy is negative, a bronchogram should be taken. At times this demonstrates an obstructing lesion in one of the smaller bronchi beyond the visual field of the bronchoscopist. A negative bronchogram, however, does not rule out a peripheral pulmonary malignant neoplasm. It is of value only when it gives positive results.

Surgical exploration of the chest is a safe procedure, and should be utilized more frequently to determine the etiology of unexplained pulmonary lesions. Physicians refer cases with unexplained abdominal disease for exploration without hesitancy, but for some unknown reason are extremely reluctant to refer patients for thoracic exploration. With modern operative technic, exploration of the chest can be performed with nearly the same degree of safety as an abdominal exploration, and with equally satisfactory results. The importance of early exploration is shown by the fact that it served to establish the diagnosis in 24 per cent of the 153 cases of carcinoma of the lung reported by Overholt.[1] This procedure should be used without delay in all cases with proved or suspected pulmonary or mediastinal tumors, unless evidence of metastasis or extension of the tumor is present or the age and general condition of the patient contraindicate an operative procedure.

Aspiration biopsy is used to secure tissue for pathologic study only in cases that are obviously inoperable. In patients in whom operation is possible, exploration is considered to be safer and more accurate.

Discussion

Rienhoff[12] has admirably expressed the need for early diagnosis in cancer of the lung as follows: "Our clinical pace must be quickened, for until recently the great majority of recognized affections of the respiratory tract have been those in which Time was felt to be the Great Healer, but with the development of methods by which patients can not only be relieved, but cured of malignant neoplasm of the lung, it may be said that Time now takes on the role of executioner." The same idea can be applied to pulmonary tuberculosis and most of the other thoracic diseases. Almost without exception, the success of treatment is dependent on early diagnosis

and the prompt application of the indicated therapeutic measures.

Routine roentgenologic examination has been proved to be a valuable supplement, but should never be considered a substitute for a carefully taken history and a methodically performed physical examination. Occasionally, the history and physical examination point the way to the diagnosis when the roentgenogram reveals no definite abnormality. This is especially true of the early stages of bronchial disease, when the only symptom may be localized wheezing. It is also quite true of bronchiectasis, in which the presence of rales may first indicate the presence of the pathologic process.

The evidence already presented shows the value of routine roentgenologic study of the chest. This is particularly important in patients in whom the incidence of pulmonary disease, especially tuberculosis, is known to be high and the threat is known to be great, such as pregnant women and diabetic patients. The early diagnosis of pulmonary tuberculosis is important for pregnant women from the standpoint of giving proper care to the mother and adequate protection to her baby. It is important in diabetic patients because of the well-known fact that these two disease processes aggravate each other.

Another group of tuberculous patients are those in their late teens and young adults. Early diagnosis is more important here than in any other age group because of the relatively poor prognosis and the tendency for the lesions to progress rapidly. Once a lesion is discovered in a patient in this age group, it should always be considered active until proved otherwise by careful clinical and x-ray follow-up. If it is active, immediate intensive therapy and prolonged bed rest are indicated. Roentgenograms of the chest should be taken at frequent intervals, sometimes as often as once a week. In such cases, tuberculosis is not a chronic disease but one that carries a great and an immediate threat to the patient.

The responsibility for the early apprehension of pulmonary disease rests largely on the shoulders of the general practitioner and the internist, since they are the first to see the patient. Their offices should and can be the greatest case-finding agencies in the entire field of medical practice. To make this possible, the limitations of the history, the physical examination and certain laboratory procedures must be more keenly appreciated, and more rigid diagnostic procedures must be applied routinely.

Summary

Mass x-ray surveys have demonstrated that intrathoracic abnormalities, neoplasms and infections are much more frequent than has ever been suspected.

The early diagnosis of these lesions is difficult because in the early and not infrequently in the moderately advanced stages they cause few or no symptoms or signs.

Serious intrathoracic lesions are great masqueraders. They may simulate the clinical picture of any of the commonplace diseases of the chest or of an entirely foreign clinical entity, such as arthritis.

History-taking and physical examination are essential procedures in the examination of all patients, but are unreliable in the early diagnosis of diseases of the chest.

Every patient with pulmonary symptoms should have the benefit of a roentgenogram of the chest.

Surgical exploration of the chest is a relatively safe procedure and should be used early to determine the etiology of unexplained intrathoracic lesions.

Routine roentgenologic examination of the chest reveals a high percentage of significant thoracic abnormalities and diseases. This percentage is so high that every physician should strive to supplement each physical examination with at least a carefully performed fluoroscopic examination.

In the light of present knowledge, some form of roentgenologic examination of the chest should be a routine procedure for every patient admitted to hospitals and clinics. It not only reveals more significant disease than any other routine procedure now in use, but also protects other patients and the hospital personnel from the unsuspected cases of pulmonary tuberculosis that are constantly present in hospitals.

A series of illustrative case reports is appended.

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References

1. Overholt, R. H. Common masquerading lung disease. *New York State J. Med.* 42:1657-1662, 1942.
2. Alexander, J. Observations on intrathoracic neoplasms. *Ann. Surg.* 114:734-752, 1941.
3. Salkin, D., Cadden, A. V., and Edson, R. C. Natural history of tuberculous tracheobronchitis. *Am. Rev. Tuberc.* 47:351-369, 1943.
4. Kallner, S. K. On prognosis of exudative pleurisy. *Acta. Med. Scandinav.* 92:549-568, 1937.
5. Trudeau, F. B. Pleural effusion: its prognosis in patients showing little or no parenchymal involvement. *Am. Rev. Tuberc.* 39:57-63, 1939.
6. Sampson, H. L., and Brown, L. Correlation of clinical and roentgenological observations in pulmonary tuberculosis. *Radiology* 22: 1-14, 1934.
7. Fellows, H. H. Significant active pulmonary tuberculosis in apparently healthy adult: study of 141 cases. *Am. J. M. Sc.* 188: 533-537, 1934.
8. Potter, H. E., Douglas, B. H., and Birkelo, C. C. Miniature x-ray chest film. *Radiology* 34:283-291, 1940.
9. Block, R. G., and Tucker, W. B. Indispensability of routine x-ray examinations of chest in general clinic. Unpublished data.
10. Hodges, F. J. Fluorographic examination of chest as routine hospital procedure. *Radiology* 38:453-461, 1942.
11. Pinner, M., and Werner, W. I. Significance of positive and negative sputum findings in pulmonary tuberculosis. *Am. Rev. Tuberc.* 18:490-496, 1928.
12. Rienhoff, W. F., Jr. Carcinoma of lung. *Surgery* 1:125-129, 1937.

Illustrative Case Reports

Case 1. J. M., a 44-year-old man, had for years had a chronic cough attributed to cigarette smoking. In March, 1942, he had blood-streaked sputum. A roentgenogram of the chest at that time showed infiltration in the right upper lobe that closely resembled a tuberculous lesion (Fig. 1*A*). The sputum was consistently negative. In spite of this, a diagnosis of pulmonary tuberculosis was made, and the patient was sent to Florida for a few months. During that time the cough persisted but the quantity of sputum became less. There was no weight loss.

During the 4-month period following the initial roentgenogram, the shadow in the right upper lobe became quite dense and rapidly enlarged (Fig. 1*B* and *C*). A diagnosis of tumor was made and the patient was referred for surgery in August, 1942, about 5 months after the first roentgenogram of the chest. Bronchoscopy was negative. Exploratory thoracotomy was immediately performed. At operation a huge tumor mass was found in the right upper lobe. No mediastinal lymph nodes were palpable. A pneumonectomy was performed. The patient made an uneventful recovery (Fig. 1*D*) and remained well until December, 1942, at which time he began to have low-grade fever and severe pain in the left lumbar area. He rapidly went downhill and died on February 22, 1943.

Pathologic examination of the surgical specimen revealed epidermoid carcinoma, Grade III, with metastasis to the bronchial lymph nodes. At the time of autopsy, metastases were found in the spleen, the left adrenal gland and the peritracheal and supraclavicular lymph nodes.

Comment. This case demonstrates carcinoma of the lung masquerading as pulmonary tuberculosis in its early stages. The persistently negative sputum should have indicated further diagnostic procedures. An exploratory thoracotomy at the time of the first roentgenogram would have settled the diagnosis, and pneumonectomy at that time would have offered a better chance of cure.

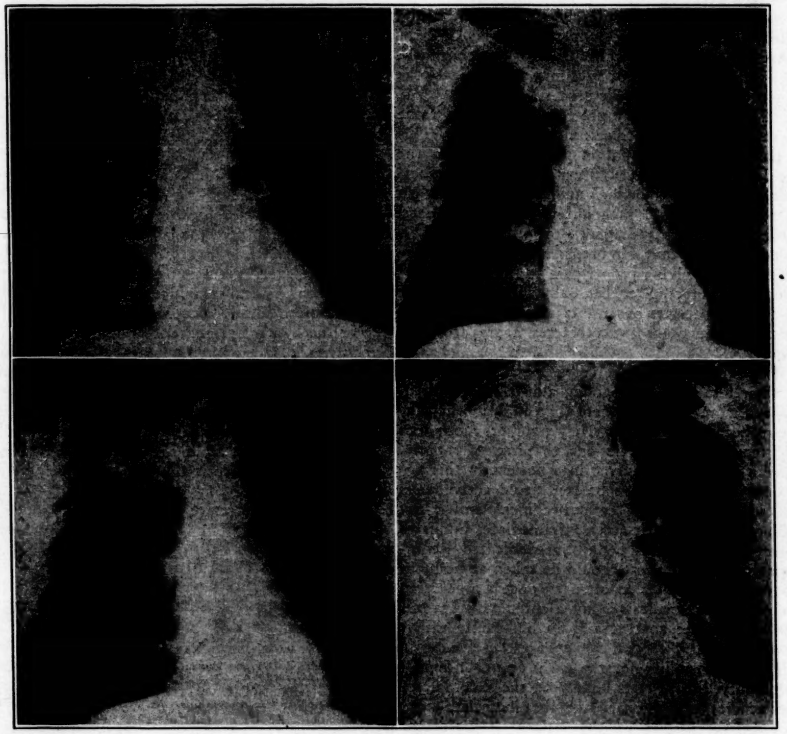
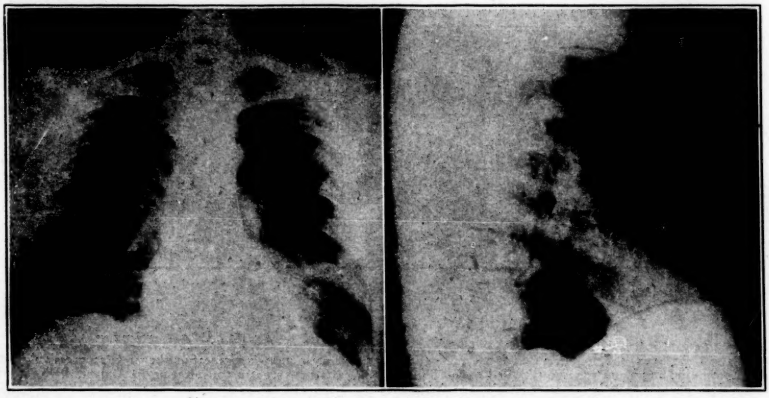


Figure 1. *Case 1.*

Case 2. G. B., a 51-year-old man, became ill in February, 1943, at which time he was treated for "grippe." He never felt well following this, and mild cough and expectoration

continued. In July, blood streaking of the sputum first occurred. This reoccurred on several occasions. Roentgenograms revealed infiltration in the left lower lobe. There had been a weight loss of 20 pounds. The patient was treated for unresolved pneumonia. He was first seen in consultation in December, 1943, about 10 months after the onset of symptoms. At that time, the roentgenogram (Fig. 2*A*) revealed an opacity adjacent to the left border of the heart. The lateral view showed the opacity to lie posteriorly in the lower lobe (Fig. 2*B*). Bronchoscopy was negative. Exploratory thoracotomy was performed and a tumor mass (epidermoid, Grade II) was found in the lower lobe, which had extended into the mediastinum around the inferior pulmonary vein. A palliative pneumonectomy was performed. X-ray therapy was used postoperatively. The patient was last seen on April 4, 1944, at which time his general condition was good and there was no evidence of further metastasis.

Pathologic examination revealed a 4-by-4-cm. tumor in the lower lobe. Beyond the tumor, the lung was atelectatic and the bronchi were dilated.



A B

FIGURE 2. *Case 2.*

Comment. This case illustrates bronchiogenic carcinoma presenting the clinical picture of grippe at its onset. The first roentgenogram of the chest was not taken until 6 months after the onset of symptoms, and the patient was not referred for surgical treatment for another 4 months. A roentgenogram should have been taken in February, 1943, the ideal time for exploratory thoracotomy. A curative type of pulmonary resection might have been performed then.

A B

FIGURE 3. *Case 3.*

CASE 3. D. M., a 26-year-old, married diabetic woman, had a chest roentgenogram in 1931 that revealed a small scar

in the apex of the right lung. The patient was not instructed to have frequent x-ray check-ups. The next film was taken in February, 1941, and an infiltration was found in the right apex. A checkup was advised in 2 months' time. The patient failed to return, and the next roentgenogram was taken in October, 1942 (Fig. 3*A*). At that time there was an exudative lesion at the right apex but no visible cavity. The patient was asymptomatic, and there were no physical signs. Crushing of the right phrenic nerve was performed, and the patient was placed on bed rest.

During the following year, x-ray films showed progressive retrogression of the lesion at the right apex, and the patient remained asymptomatic. She was x-rayed every 3 months during this time. In January, 1944, a roentgenographic checkup revealed a definite spread of the lesion (Fig. 3*B*). The patient still had no symptoms, and the physical examination was entirely negative.

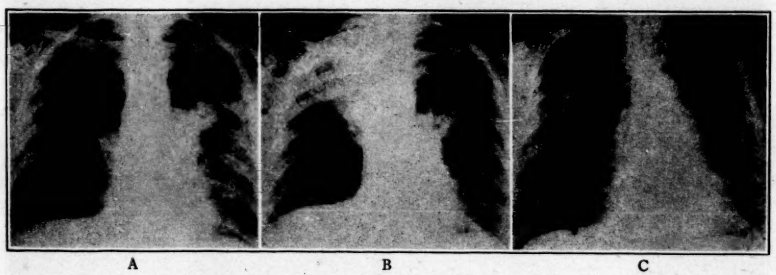


FIGURE 5. *Case 5.*

Comment. This case illustrates a silent tuberculous lesion occurring in a young diabetic patient. It demonstrates the inadequacy of the history and physical signs in establishing the diagnosis and also in evaluating the course of the disease process.

CASE 4. V. S., a 51-year-old, married woman, had an acute illness in May, 1943, and was told that she had pneumonia. Following the acute episode she failed to regain her strength,

A B C

FIGURE 4. *Case 4.*

and cough and expectoration became progressively worse. She was first seen in consultation in August, 1943, at which time the outstanding symptoms were marked cough, expectoration of 8 to 10 cc. of mucopurulent sputum daily and wheezing in the left side of the chest. A roentgenogram of the chest (Fig 4*A*) revealed a dense opacity in the midzone of the left lung adjacent to the hilus. The patient's age, the clinical picture and the roentgenogram all pointed to a diagnosis of bronchiogenic carcinoma. This was especially true because the sputum had been consistently negative on repeated examination by the concentration method. Bronchoscopy revealed edema and thickening of the mucosa throughout the left bronchial tree. The mucosa was also slightly inflamed and granular in appearance. No evidence of tumor was found. Bronchoscopy indicated that the process was probably due to tuberculosis. A few weeks later the sputum was proved by culture to be positive for tubercle bacilli.

The patient has been followed by bronchoscopy since then. The process extended into the trachea and eventually ulcerated. The upper portion of the right main bronchus also became involved. In October, 1943, atelectasis of the right upper lobe occurred; it disappeared within a few weeks (Fig. 4*B*). In recent months the patient has improved, the ulceration in the bronchus has disappeared, and the infiltration in the left lung has cleared slightly. There is now, however, a suspicious cavity in the apex of the left lower lobe (Fig. 4*C*).

Comment. This case illustrates tuberculosis masquerading as cancer of the lung. It also demonstrates the importance of rigid study of the sputum before deciding that it is negative. Bronchoscopy was helpful in establishing the diagnosis.

CASE 5. F.R., a 55-year-old man, "caught cold" 8 weeks prior to the time he was referred for treatment. This illness subsided, and he felt well. One month later he had several hemoptyses, a low-grade fever and some malaise and had lost 15 pounds. A roentgenogram of his chest taken at the first examination on June 16, 1943, showed density in the region of left hilus (Fig. 5). Bronchoscopy was negative, and bronchograms revealed no abnormality. Exploratory thoracotomy was performed and a small tumor was found in the left upper-lobe bronchus. Pneumonectomy was performed. The patient is asymptomatic and well.

Pathologic examination of the surgical specimen showed a small Grade III epidermoid carcinoma in the left upper lobe bronchus. All the lymph nodes were normal.

Comment. Only 8 weeks intervened between the onset of symptoms and the time at which definitive surgical treatment was performed. This case shows the value of early explora-

tory thoracotomy in establishing the diagnosis and thus affording a good chance of cure.

Cases 6, 7, 8 and 9. These 4 patients had intrathoracic lesions that produced absolutely no symptoms. In all the diagnoses were made by routine roentgenograms of the chest.

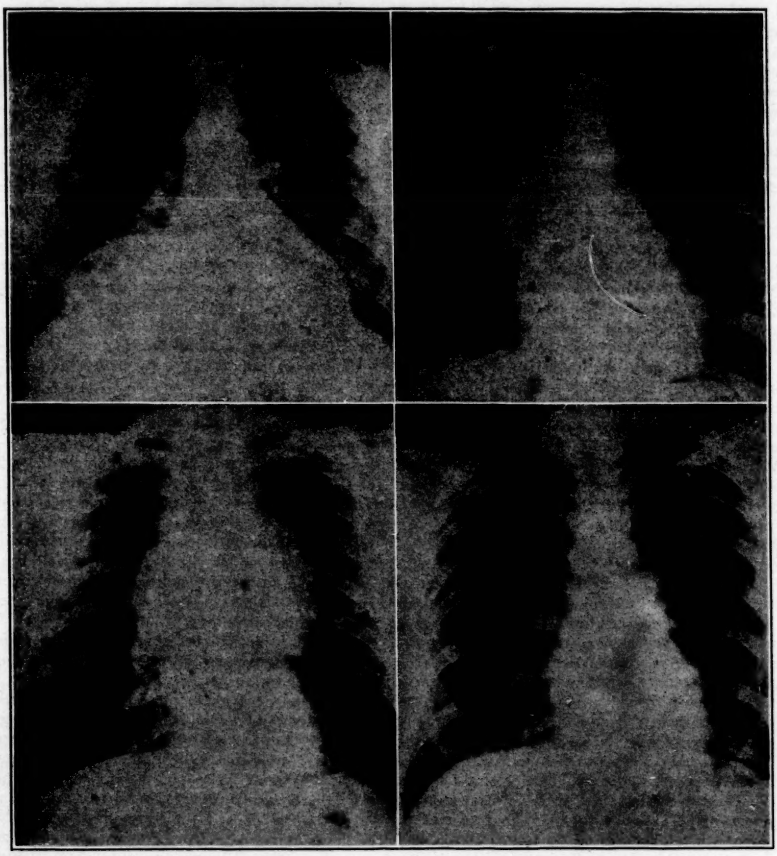


Figure 6. *Cases 6, 7, 8 and 9.*

Figure 6*A* shows the chest roentgenogram of a 16-year-old boy (Case 6) with a diaphragmatic hernia, which was discovered in a school survey. Figure 6*B* shows the chest roentgenogram of a 22-year-old student nurse (Case 7) with a mediastinal cyst into which hemorrhage had occurred; physical examination was negative, as well as a roentgenogram of the chest taken two years previously. Figure 6*C* shows the roentgenogram of a 20-year-old man (Case 8) with a huge mediastinal tumor, which proved to be an adenocystoma; this was discovered while the patient was on active duty in the Marine Corps. Figure 6*D* shows the roentgenogram of a 23-year-old man (Case 9) with moderately advanced pulmonary tuberculosis involving the left upper lobe; there was a 3-cm. cavity at the level of the anterior second rib, without physical signs or symptoms. The first three patients have been operated on successfully and are in perfect health; the tuberculosis of the last patient is under control with pneumothorax.

CHALLENGES TO MEDICINE*

VICTOR JOHNSON, M.D.†

CHICAGO

THROUGHOUT the country, many hundreds of medical students are graduating this month. Nearly all of them automatically become Army and Navy officers, scheduled to be assigned to duty in the jungles of Burma, the deserts of Africa, the snows of Alaska, the mountains of the Balkans or the interior of Germany or Japan. They, and you, can embark for duty in a far corner of the earth supremely confident in yourselves, and your complete competence to meet every situation.

If your medical education has been hastened, remember that the accelerated program has already provided new medical officers for many thousands of our fighting forces. If your classes have been crowded, know that there will be fewer doctors for the civilian population after the war than before the war, despite acceleration and increased enrollments. If your teachers have been reduced in numbers, be sure your medical education is still far superior to that obtainable anywhere in the world, even in peacetime. If you have been beset from day to day with numberless uncertainties, recall that you share doubts with the entire civilized world.

We have supreme confidence in you, in your ability to discharge your obligations not only in this great conflict, but in the equally difficult readjustments to be made after the war.

Medical students, medical faculties, medical scientists — all of American medicine — may well hold their heads high in any accounting that is held of contributions to winning the war. Thousands of men who would have died in the last war will return to useful and happy lives because of plasma, penicillin, surgery, air evacuation of the wounded and because of the medical officers — because of you. We know that as you have met your responsibilities in the past you will carry on the proud tradition of service in the immediate future during this conflict, and we know that you will likewise accept with resolution the challenges of the future in the world of peace, which will even transcend in importance the tasks of today. The problems to be met are those of education, adjustment to civilian life and expansion of the effectiveness of medicine in a changing world.

EDUCATION

The last war was won by food. "Food will win the war." It was won by the United States, with her fresh, strong forces. The war was won by the French holding fast before Paris, by the British blockade of Germany, by the defection of the German people, by the German sailors at Kronstadt, by the American skilled worker — by everyone who participated.

This war will see fewer such credit lines. Perhaps we have become mature. German is still taught in our colleges, and many medical schools still require it for admission. Wagner still fires the pulse, and we continue to trust Goethe, who could say to a world less sick than ours, "Thou ailest here, and here, and here."

We have reached maturity, perhaps, and we shall not say, "Brains won the war, education won the war." Education has done its stint. Colleges, universities and professional schools have made available their entire educational resources, and many many thousands of men will be better citizens in peacetime for the education they received for war.

I need not recount to you the many Army Specialized Training and Navy programs. You are familiar with the numerous projects in operation: radar and radiography; parachute physiology and parasite detection; planes and plasma.

Education for war has been universal, but the education of soldiers for peace has also taken place. The Armed Forces Institute offers correspondence courses in any subject and at every level, to every soldier or sailor with leisure for study. An educational project under the Army Education Branch is planning to serve the demands and intellectual curiosities of thousands of soldiers in the interim between the surrender of Germany and that of Japan. It is anticipated that many thousands of men will spend weeks to months in Europe before the crowded transport facilities of that time will be able to carry away the last of our armies to America or the Pacific. How can that time be most profitably utilized? For many, it will be a period of formal organized education. Scores of courses are now being developed in all the fields of learning, from the level of the high school to advanced university work. Textbooks have been selected and outlines prepared for courses varying from one to several months. Wherever possible, physical educational facilities in Europe will be used, including the modest school buildings of the small cities and the laboratories of the great universities. The instructors will come from the officers and men. Here a private who has had a year of college will give instruction in high school biology; there a Ph.D. will give college instruction.

Some of the educational projects and programs of this war approach the fantastic. Vice-Admiral Ross

*Convocation address, delivered at Boston University School of Medicine, Boston, September 22, 1944.

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McIntire, Surgeon General of the Navy, tells of clinics held in midocean. In a convoy not in trouble, or in a task force on a long journey, the medical officers in the many vessels of the fleet meet at a stated time on one of the larger vessels. Here are assembled, under the direction of competent medical officers of higher rank, selected cases of general interest and stubborn diagnostic problems encountered in any large body of men subject to illness or injury. A clinic is held, especially for the instruction of the younger officers whose hospital training has been curtailed. This is medical education that may be characterized as unconventional, at least.

At the University of Edinburgh, in Scotland, an event unique in medical annals is occurring. When Poland was overrun, thousands of Polish officers and men and civilians were evacuated to England. Many of these had been medical students. Among the medical officers were a goodly number of teachers from the Polish medical schools. The University of Edinburgh provided classroom, laboratory and clinic facilities, and there was set up the first medical school in the history of any country in which citizens of the country taught their fellowmen in their own tongue in a foreign land. The Polish Medical School of Edinburgh has graduated two classes of future Polish doctors, and has made a respectable contribution in research. This amazing experiment in international medicine will yield incalculable dividends in the rebirth of medicine and education in Poland, which the Germans boast has now been reduced to an intellectual desert.

In the midst of our efforts here at home to produce competent medical officers at the maximum rate, consistent with high quality, we are deeply concerned with your future continuation training. Despite the wartime educational efforts mentioned, we may be sure that when the balance is struck, the debits will far outweigh the credits, in all fields of education. In medicine, this will be reflected in a lower quality of medical care after the war, unless the challenge is met resolutely and wisely.

Recent graduates are keenly aware of their deficiencies. They have been compelled to complete their training under unfavorable conditions. Teaching staffs have been depleted while their teaching loads have been increased and the tempo of education hastened. The internship has been necessarily reduced to an inadequate nine months, a period barely sufficient to make the discerning student aware of the medical problems he will face in the practice of medicine but utterly insufficient to do more than start him on the way to their solution.

Medical educators are likewise keenly aware of the necessity for providing, after the war, continuation courses and advanced hospital training not only for new generations of medical graduates but for the returning medical officers as well.

We already know something of the magnitude of the problem. It is now nearly two years since the Council on Medical Education and Hospitals of the American Medical Association anticipated the problem to be faced, and set about to meet it, collaborating with hospitals, American boards in the specialties, medical schools and, most recently, the Committee on Postwar Medical Services. This committee was established by the American Medical Association, in collaboration with the American College of Physicians and the American College of Surgeons. Representation is also included from the Association of American Medical Colleges, the American Hospital Association, the Federation of State Licensing Boards of the United States, the Catholic Hospital Association, the Procurement and Assignment Service, the Advisory Board for Medical Specialties and the Veterans Administration.

A major project of this committee has been a study of the postwar educational desires of medical officers, which is being conducted in co-operation with the surgeons general of the Army, Navy and Public Health Service. Questionnaires on the postwar educational desires of medical officers have been sent to all physicians in all branches of the Service. Returns are now being received at the American Medical Association headquarters in great numbers. Transferred to punch cards, this information will be systematically analyzed and given wide publicity, so that each state medical society, each medical school and each hospital will be made aware of its responsibilities.

Analyses of the first 1000 returns have been published by Lieutenant Colonel Harold C. Lueth[1,2] and the Council on Medical Education and Hospitals.[3] The report of the Council indicates that considerable expansion is required in providing facilities in all fields, and for periods ranging from several weeks in refresher and review courses to many months in internships and residencies.

Of the 1000 officers, about 20 per cent indicated that they planned no further postwar training. Eighty per cent — mainly younger officers — desired some additional education. About one third of those who replied desired six months or less of training, and nearly half desired hospital training of six months or more.

To meet the requirements of returning medical officers for additional training is a serious responsibility that will require the continued joint efforts of the Committee on Postwar Medical Service, the Council on Medical Education and Hospitals, the surgeons general of the Army, Navy and Public Health Service, hospitals approved for internships and residencies, the American boards in the medical specialties, medical schools, state licensing boards, the Veterans Administration, foundations, county and state medical societies and every institution capable of providing advanced training to physicians.

On the basis of the estimated demands for educational opportunities, the Council on Medical Education and Hospitals of the American Medical Association is already engaged in a ten-point program aimed at filling these needs. Internships, residencies, fellowships, graduate externships, and review and refresher courses are being developed to meet these needs. This is a solemn obligation we owe to the young physician now at war, who has given so much, so freely and so well. But there is also the added obligation to ensure that a high quality of medical care will be maintained in this country, lest the health of the Nation should suffer for years to come. You who graduate today may count on us who lay these plans to do our utmost for your continuation training after the war.

Adjustment to Civilian Life

Those of you who remember Remarque's great novel of the last war, *All Quiet on the Western Front* will recall with deeper feeling his novel of the peace that followed, *The Road Back*. War still poisoned the returned German soldier, even in the green meadows and wooded hills of his childhood. Here he did not relive the games of school picnics but, crouching behind a tree, noted at once what a perfect defense could be made there. He saw on that hill an artillery battery, and in that grove two machine-gun nests. There were snipers in the shrubbery. There was a perfect trap, with withering crossfire.

We escaped the Hun hatred of the last war that included an unreasonable hatred of German literature and music. Perhaps we shall be sufficiently civilized also to avoid the psychologic perils of the coming peace. Perhaps Remarque's character will be supplanted with the young infantryman I recently met on a train from the South. He was en route to Fort Sheridan to be discharged. The "fruit salad" on his left chest included three combat stars. I said: "Well, the war is over for you. And now what will you do?" He chuckled his response: "I'm going back to the farm. I'll rest awhile, maybe two weeks, with breakfast in bed every morning. Then I'll go to work. We have a fine farm. We raise corn and hogs."

Every returning soldier will face his own peculiar problem in his adjustment from a life of hardship where every decision was made for him to a life less hard but requiring him to do the deciding. Medical officers will face their own special problems.

When the war ends, thousands of physicians will have known no other form of medical practice than that of completely socialized medicine. The group practice of medicine is not socialized medicine. Neither is salaried medical practice, or hospital or sickness insurance. Socialized medicine is a system in which all doctors are paid from tax moneys, no patient pays his doctor directly, and all patients are provided with free medical care. This form of practice is necessarily the rule in the armed forces. Army and Navy medicine is completely socialized, and is the form of practice of one third of the active physicians of the United States today.

In the armed forces, everyone who needs medical care must get it. The economic status of the patient is never even considered. The only criterion for the best of treatment is the need of the patient. Every resource of medical science must be employed when available and indicated: plasma or penicillin, surgery or psychiatry, regardless of cost. No obstacle can be too great to surmount, whether it may require eight natives to carry a single wounded soldier to a medical station in the Owen Stanley Mountains, or a fleet of air transports to carry the injured from the fields of France to a hospital in England.

There is no doubt that wartime experiences of physicians and of patients will stimulate increased efforts toward the provision of improved medical care for everyone, but without that necessary total regimentation that must obtain in the armed forces. Such regimentation is complete, in every phase of the life of both the doctor and the patient. The patient is ordered to appear before the doctor, and the doctor in turn may order the patient to the hospital. This has not been possible in civilian life. It is very disturbing to everyone who is interested in providing the best medical care for everyone to examine certain hospital statistics in this country. If the various states are arranged in the order of number of available hospital beds per 1000 people, it is seen that the same order is followed in the percentages of those beds that are occupied. But the correlation is the reverse of what we should expect. In general, those states having the fewest hospital beds have the lowest rates of occupancy of those beds. States with most beds use them most.

Why this is true we do not know. It may be that in those states that lack the money for hospital beds the people lack the money to use them. It may be that some of these hospitals do not provide a high grade of medical care. It may also be that the people do not sufficiently demand medical care even when it is available, because of ignorance, superstition, prejudice or sheer inertia. Even among the more fortunate classes of people, full use is not made of available medical facilities. I dare say that virtually everyone here would subscribe to the principle of an annual physical examination for everyone. Yet extremely few seek such examinations, even in normal times, unless compelled by an insurance, employment or other requirement, or unless frank illness sets in.

The demand for medical care in civilian life is different from the need for it. One of our major goals must be educational: to inform people of the facilities available and the necessity for using them. Such a program of education of the public for the prevention of disease should center about the hos-

pital. The educational responsibilities of the hospital become twofold: the training of medical graduates and the informing of the public.

These considerations, these consequences of war and the practice of medicine in the armed forces, carry us to the third of the major challenges that must be met by the medicine of tomorrow.

Expanding the Effectiveness of Medicine

The drives that have carried American medicine to its present high position will continue, and will carry it far beyond the present frontiers of service to mankind. The first of these undeniable urges is the spirit of scientific inquiry. It made Von Mering and Minkowski wonder why their operated dogs voided urine containing sugar, and it kept Banting and Best from quitting when their first experiments on diabetic dogs were inconclusive. It gave the world insulin.

This urge impelled Whipple to try empirically all the edibles that occurred to him in the treatment of dogs he had made anemic, and it stimulated Minot and Murphy to extend the observations to human beings. And today, we have the liver treatment of pernicious anemia.

But though we can now control these formerly fatal disorders, we still do not know their basic cause. We cannot prevent them, and when treatment is suspended, the diabetic or the pernicious anemia patient is as seriously ill as ever. The first causes are still unknown and uncontrollable. The successes of the past provide ample hope, however, that this challenge will also be met, not only in diabetes and pernicious anemia but in hypertension, infantile paralysis and cancer, as well as in meeting the problems of a more equitable distribution of medical care.

Scientific experimentation not only must provide the solution of the problems of control of disease but also must guide us in determining the forms that medical practice shall take in the future. Medical science plans experiments, measures results and plans further experiments, to the end of greater and greater understanding of man in health and disease. This same spirit must direct us in solving the equally complex problems of man's relation to his fellows and the place of medicine in a changing social and economic structure.

Changes will come. You will be involved in them. In part, you will direct them. In so far as you exert influence, you should be impelled by the impartial spirit of inquiry that served you so well in your pursuit of the truth in the medical sciences.

We must recognize the extreme complexity of the problem. To state that doctors are attracted to cities because they can make more money there is a gross oversimplification. Cities provide the essential tools for the practice of medicine, which have been many times multiplied — x-ray equipment, electrocardiographs, other laboratory facilities, consultation services and, above all, hospitals.

The economic level in many rural communities, and in many counties in the United States, makes it impossible for those communities, unaided, to provide either doctors or the necessary facilities for doctors to carry on their work. It seems clear that hospital facilities will be lacking or will remain at a substandard level in many rural communities in the United States unless there is increased state and federal aid for hospital construction and equipment, the organization of clinics and the development of traveling clinics, to be operated under local control.

But even if all this were provided for the country physician, problems of distribution of medical care would remain. The doctor demands, in common with his civilized fellows, a life with stimulating social, intellectual and cultural contacts. Good schools, homes, churches, music and comradeship may loom as large in his selection of a community to practice in as income, x-ray facilities or hospitals.

The distribution of medical care is only one aspect of the larger problem of elevating the general economic level of large numbers of our population in peacetime. If unemployment is kept as low as now, and production is maintained at the present level after the war and dedicated to the welfare of the people, the problems of distribution of medical care will be greatly diminished.

Difficult though these problems are, they must be attacked. Here, as in science, experimentation provides the most promising approach. New experiments are being planned almost daily, and the reports are rapidly accumulating. The California Physicians Service, under the auspices of the San Francisco County Medical Society, is rapidly increasing the scope of its services. About a year ago, the rate of growth of the Service was about 1000 new members a month. Today, 6000 members a month are being added, although the net gains are less than these figures indicate because of the turnover in employment.

The Prepaid Medical Service in Connecticut is operating on a cash-indemnity basis under the auspices of the Connecticut State Medical Society. The Committee on Medical Economics of the Kansas Medical Society is developing a prepayment plan for certain medical services. In Michigan it is reported that one person in nine is covered by Michigan Medical Service certificates, and one in five by those of Michigan Hospital Service.[4]

The Missouri State Medical Association has unanimously adopted a plan of prepayment for medical and surgical care in hospitals, to be known as Missouri Medical Service, Inc.

In Nebraska, the House of Delegates of the Nebraska State Medical Society has adopted a resolution providing for the study of insurance plans and organization of such a plan for Nebraska.

Numerous other plans have been in operation, for varying periods, on a statewide or county basis, under the auspices of the medical profession. This is true in Colorado, Massachusetts, New Jersey, New York, Pennsylvania, Utah, Texas, Kansas, Delaware and North Carolina. These plans must be looked on as experiments in the more complete and equitable distribution of medical care.

As in the fields of the more exact sciences, these experiments in medical sociology are likely to turn out to be poorly conceived, in some instances, and often the results may be difficult to interpret. But the all-important fact remains: experiments in the distribution of medical care are in progress, and we may hope that they will be as fruitful as those that provided insulin for diabetes, liver for pernicious anemia and penicillin for infections.

We are impelled to continue experiments of this kind, as well as experiments in carrying patients to the doctors with adequate modern facilities, recalling the air transport of the wounded in battle, instead of struggling to provide doctors and hospitals for every community. We must experiment with traveling clinics and the closer integration of diagnostic, hospital and expert consultation services. Above all, we must experiment rather than design blueprints in the skies; we must follow the methods of science, not the promptings of the emotions.

* * *

You who are graduating are heirs to a glorious heritage of the past. And you face a challenge unequaled in the past. We will afford every help in the furtherance of your educational aims. We will assist you in your adjustment to civilian practice. And we enlist your enthusiastic endeavors to make the art and science of medicine, which you have so well learned, available to all who need it.

We know that you will serve your fellows selflessly. Despite your trials, you, and all of us who have remained at home, have little justification for complaint. All of us here owe so much to our friends and allies. A recent medical graduate in Britain[5] has put it well:

> It is hard for my immediate generation to forget that their services have been paid for already, by the bodies of men no stronger and brains no less trained. It is difficult to say that we deserved it, even when it was part of a well-laid, state-planned calculation. We were reserved, held out of the line, while Jock (who was trained as a diplomat) took his platoon behind the enemy lines to destroy thirty-six aeroplanes and was killed by a bleeding artery on the way home; and Michael died when he led his company on to the last objective at El Alamein (he had passed his final examination before ordination); and Melvin bombed the dam when he could have stayed comfortably in the RAF in America. If such people had not given up their jobs and gone to war we should not be doctors now. It is my salutary misfortune to remember how much they entertained the world without trying, and helped the world without the special equipment of medicine.

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References

1. Medicine and the War. Results of pilot questionnaire to physicians in service. *J. A. M. A.* 125:558-560, 1944.
2. Lueth, H. C. Future educational objectives of medical officers. *J. A. M. A.* 125:1099-1103, 1944.
3. Johnson, V., and Arestad, F. H. Educational facilities required for returning medical officers. *J. A. M. A.* 126:253-257, 1944.
4. Michigan medical service. *J. Michigan M. Soc.* 43:623, 1944.
5. In the near future (licentiate of the college speaking). *Lancet* 2:262 1943.

MEDICAL PROGRESS

INSECT VECTORS OF DISEASE

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BOSTON

SINCE December 7, 1941, all Americans have become conscious of war — for Japan had suddenly burst America's bubble of isolationism. Although many persons considered the United States as self-sufficient and apart from the rest of the world, and therefore safe from the conflagration of war, public-health officials, however, have been deeply concerned, fearful of the involvement of the country in a global war, with its inevitable rise in infectious diseases and death.[1] Since time immemorial, wars have been attended by pestilence and death.

Under war conditions, many of the safeguards of the health of both the military and civilian populations are abandoned.[2] The crowding of people, the primitive environmental conditions and the lack of personal hygiene are all conducive to the spread of infectious disease. The concentration and movement of large groups of population — civilian and military — and the limitless hardships, with fatigue, general malnutrition,[3] famine and exposure, and the lack of medical care experienced by civilians and some armies, provide the fuses for the explosion of epidemics.

More soldiers have invariably been killed by infectious disease than by bullets in all wars prior to the Franco-Prussian War of 1870, when the German Army reported an excess of deaths from battle.[4] In the Russo-Japanese War of 1904, both sides maintained the high proportion of deaths from wounds. In spite of the toll of influenza, the American Expeditionary Forces of 1917–1918 reported fewer deaths from sickness than from battle; although for the entire United States Army, at home and abroad, the deaths from disease were more numerous. During and after World War I the usual war diseases were highly prevalent in eastern Europe.[5] There was an epidemic of typhus fever throughout Russia, Poland and Serbia. Fortunately, the disease did not spread to western Europe. Malaria was victor in Macedonia,and decimated the opposing armies to a tragic ineffective fraction.[6] Although it had previously disappeared from most of Europe,[7] malaria returned with the soldiers and evacuees to central, eastern and northern Europe to ravage the population. The United States Army, fortunately, escaped any extensive involvement of insect-borne disease, as the zone of military operations did not extend into areas where these diseases were endemic.

This global war is much more hazardous to civilian health than any in history.[8] The civilian population is being exposed to the potential hazards of disease throughout the fighting zones and in those areas to which the troops are moved. This risk of infection will probably not reach its peak during the actual warfare, but rather in the period of population readjustment following the cessation of hostilities.

Large numbers of nonimmune susceptible persons are being concentrated in areas where insect-borne diseases are prevalent. With battles raging in all parts of the globe and principally in the tropics and subtropics,[9] new hazards face the fighting men. Under battle conditions, they are being exposed to the added risk of the limitation of known effective control measures, unavoidable in the face of combat. To the classic list of military diseases, such as dysentery, typhoid, paratyphoid and typhus fevers and malaria,[10] the military personnel have now the added risk of an entire galaxy of tropical diseases,[11] many of which are insect borne. The problem presented by latent unrecognized infection[12] and the carrier state among the military personnel cannot be fully appreciated. The similar problem presented by refugees in mass emigration from war-devastated areas or because of postwar treaties cannot be fully anticipated. The danger to the civilian population of the United States does not arise from the recognized and treated cases but from those with latent infections, from carriers and from refugees, all of whom may bring infections back to their homes and thereby infect the indigenous arthropod vector.[13–15]

In the present war, arthropod-borne diseases have already played an important role.[16] Typhus fever has reached epidemic proportions throughout eastern Europe and has extended into all of Europe, North Africa and Asia Minor.[17, 18] The hazards of insect-borne diseases have been intensified by the crowding in bomb shelters and concentration camps. Scabies, impetigo and lice infestations are acute problems. The incidences of typhus fever[19] and of relapsing fever, which had been decreasing during the decade preceding the war, have again increased in Poland, Russia and Romania, which are the principal European foci of these diseases. Similarly, malaria has again taken its toll, but to a far greater degree.

Man has, to a large degree, been able to control those infections for which he has been able to develop an artificial immunity by inoculation, such as smallpox and typhoid fever, and those diseases against which isolation is an important factor. Under wartime conditions, however, the control of infectious diseases is of military importance only

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in so far as these interfere with the task assigned to any military unit[20]; and therefore, the main purpose of infectious disease control during wartime is guided by the principle of keeping as many men physically fit and in fighting trim as possible. In other words, the importance of a disease from a military point of view and the value of disease-control measures are determined in wartime by their effect or noneffect on the available fighting strength of a force.[21] Insect-borne diseases are perhaps the most difficult and among the most important diseases with which military personnel are afflicted. For example,[22] malaria was one of the main forces that defeated the American troops at Bataan[23]; it again assumed major importance in the Solomons and on Guadalcanal.[24]

A disease is classified as insect-borne[25] when a blood-sucking insect is the sole or the usual agent by which the infecting organisms are transmitted from one patient to another. This transmission may be from man through vector to man, from animal through vector to man, from animal through vector to animal and from man through vector to animal. The disease may have a wide host selectivity, and the vector may spread the disease from one genus of animals to another. On the other hand, many insects feed only on certain closely related species of the same animal, whereas other vectors are not so finicky about the type of blood they suck. Many insects prefer certain hosts and fly many miles to obtain this favorite blood, rather than bite some other species that may be near by. Occasionally, this zoophilic deviation of the insect vector can be used as a control measure in malaria.[6] The importation of cattle may deviate the mosquitoes from man and thereby reduce the incidence of bites of man.

An insect-borne disease may be transmitted by only one genus or, in some instances, by only certain species or merely one species of insect. Other insect-borne diseases are spread by several different insects, entirely unrelated and of different genera. Dengue,[26] for example, was thought to be transmitted naturally by only two species of Aedes mosquitoes, *Aedes aegypti*[27] and *A. albopictus*,[28] but *A. scutellaris hebrideus* has been recently incriminated.[29] Malaria is transmitted by some twenty species of Anopheline mosquitoes.[30] Relapsing fevers are transmitted by both lice and ticks; and typhus fever by lice,[31] fleas[32] and mites.[33]

Classification of Vectors

Many so-called "insect-borne" diseases are not transmitted by insects but by other genera.[34, 35] The true insect is a hexapod and belongs to the class Insecta, such as the mosquito, fly, bedbug and louse. Other diseases are transmitted by the class Arachnida, which are eight-legged creatures, such as mites and ticks. The phylum including both the insects and the arachnids is Arthropoda — animals with jointed legs.

The class Insecta is composed of about twenty orders, of which five are incriminated directly or indirectly in the transmission of disease. These orders are Diptera (flies and mosquitoes), Anoplura (lice), Siphonaptera (fleas), Hemiptera (bugs) and Orthoptera (roaches). To these may be added a sixth order, Hymenoptera (wasps, bees and ants), some of which bite or sting man and are a source of discomfort. Perhaps some future research may relate these insects to disease transmission.

The class Arachnida is composed of eleven orders, of which only one, Acarina, comprised of ticks and mites, is known to transmit disease. The orders Scorpionida (scorpions) and Araneida (spiders) cause discomfort by biting man but have not been incriminated as vectors of disease.

Arthropods may affect the health and well-being of man in several ways: by conveying disease, by invading the tissues of man, as in scabies, by inoculating poisonous substances,[36, 37] such as in the bite of the black-widow spider, bee or wasp, and by being pests to man, such as bedbugs. This paper, however, is limited to a brief discussion of the diseases conveyed by arthropods and those of the arthropods themselves.

Diptera

Mosquitoes

The most important arthropods responsible for the transmission of disease are the true insects, in particular mosquitoes and flies. Mosquitoes transmit filariasis,[38] malaria,[7] leishmaniasis,[39] tularemia,[40] yellow fever,[41] dengue fever,[26] Rift Valley fever, equine encephalomyelitis,[42] Japanese encephalitis[43] and St. Louis encephalitis.[44] Not all mosquitoes are vectors of disease; moreover, only the female is dangerous, as the proboscis of the male is not sufficiently rigid to penetrate the skin and hence it aesthetically lives on plant juices. Three mosquito genera are of special importance as vectors — Anopheles (malaria), Aedes (dengue fever, filariasis and yellow fever[41]) and Culex (filariasis, equine encephalomyelitis, Japanese encephalitis and St. Louis encephalitis).

Not all species of any genus are capable of transmitting disease, nor are all such species of equal significance as vectors. The important vector in any particular geographic area may be missing or unimportant in some other location. For example, in the United States by far the most important vector of malaria is *Anopheles quadrimaculatus*[45]; in Italy, *A. maculipennis labranchiae*; and in Brazil, *A. gambiae*.[46] Although all mosquitoes develop by complete metamorphosis and the life cycle consists of eggs, larva, pupa and adult, all species do not breed under similar conditions.[47] Some species have only one brood a year, whereas others continue to breed throughout the year. Some lay their eggs in water-filled tree holes, and others prefer collections of water in tin cans and cisterns; still others breed in ponds, marshes, hoof-prints, puddles,

warm sunny ponds, cool running streams, or even brackish water.

The eggs of Anopheles are deposited singly in batches of forty to one hundred on the surface of the water and are provided with floats.[48] Aedes eggs are also laid singly in lots of twenty-five to fifty; they sink to the bottom, or when laid on the side of water receptacles, they may lie dormant for months, hatching only on favorable conditions of temperature and moisture.[49] Culex eggs are deposited on the water surface in rafts of one hundred to four hundred.[50] In twelve to seventy-two hours the eggs hatch into larvae, which are actively mobile and feeding cylindrical organisms; they vary in color from gray, yellow or red brown to dark brown or black. Developing by repeated moulting, the larvae pass through four instars or forms, increasing each time in size. Under favorable conditions, the larval stage lasts about ten days. Mosquito larvae must breathe through a tube at the posterior end of the body. All mosquito larvae except those of Anopheles have a distinct siphon tube, by which they attach themselves to the surface of the water, the head being suspended downward. Since all species of Anopheles have only a tubeless opening, they lie parallel with the surface of the water, their heads being just under the surface. This position makes them more susceptible to poisoning from small particles that may float on the surface of the water. From the fourth instar, the larva develops into a comma-shaped pupa, which is a resting, floating stage during which a complete metamorphosis from larva to adult transpires. The nonfeeding pupae are supplied with two breathing trumpets on the dorsum; they are actively motile, being supplied with two paddle-shaped appendages. Locomotion is accomplished by rapid flexion and extension of the abdomen.

In about twenty-four to seventy-two hours the pupal case splits along the dorsum and the adult or imago gradually emerges, and as soon as the exoskeleton hardens the mosquito is ready for flight and food. Temperature and humidity affect the biting activities, mosquitoes usually being more active in hot humid weather. The flight range varies with different species; the flight of *Anopheles quadrimaculatus* is usually less than a mile, whereas *Aedes sollicitans*, a salt-water species, has been found fourteen miles from the nearest salt water.[51] Moreover, mosquitoes, as well as other arthropods, may be transported by motor vehicles, ships and airplanes. Sailing vessels were responsible for transporting *Aedes aegypti* to Boston and other American ports from South America,[52] thereby causing short-lived local outbreaks of yellow fever. The aeroplane was probably responsible for the importation of *Anopheles gambiae* to Brazil from Africa, thereby causing an extensive and severe outbreak of malaria.[46]

The adult female mosquito, under favorable conditions, usually lives as long as three months. Fortunately, however, many succumb to the vicissitudes of their environment — natural enemies, adverse weather and other unfavorable conditions. Hence, the normal life expectancy of a female adult mosquito is probably two to four weeks.[53] Certain species of mosquitoes, however, hibernate throughout winter as adults. Thus, Culex and Anopheles, in temperate zones, survive as gravid females and some species of Anopheles hibernate as larvae. Aedes usually survives from one breeding period to another as eggs. In colder regions nearly all species survive winter as eggs, but in the tropics and subtropics unfavorable periods may be survived by retarded or slowed development.

The biting, breeding and flight habits of mosquitoes vary from place to place and from one species to another. It is therefore absolutely necessary to conduct a mosquito survey or to have available detailed information relative to the particular insect-borne disease, its vectors and the ecologic factors that are likely to bear an important role in the determination of effective and efficiently employed control measures.[51] These factors may also influence the location of camps or semipermanent institutions, such as airfields and hospitals, in relation to other population groups or settlements in the general vicinity. Similarly, the success of a military campaign may be adversely affected by lack of true evaluation of the vectors and their habits. The identification of mosquitoes is a difficult but necessary task.[54] It is usually accomplished by a key prepared for certain geographic sectors.[55, 56] Such identification into species is performed with a dissection microscope and may usually be done on both larvae and adults. Mosquito-control measures must be guided by preliminary identification surveys,[57] as well as by periodic catches of both larvae and adults to evaluate the effectiveness of applied measures. Control measures must be selective and applied only to those species that are vectors of the disease for which control is being exercised, since money, time and labor might otherwise be expended lavishly for controlling harmless mosquitoes. Similarly, nuisance mosquitoes must be controlled if effective results are to be expected. Mosquito-control measures are usually classified as temporary or permanent.[58] Temporary measures are the application of larvicides,[59] spraying with insecticides[60] and the use of insect repellents.[61] Permanent control measures include flooding,[62] drainage and the construction of ditches, dams or special siphon apparatus[63] for the automatic changing of water levels. The choice of method[64] depends on the availability of time, labor and funds and the desired result.

Flies

Whereas all true mosquitoes belong to the family Culicidae, suborder Nematocera of the order Diptera, flies are members of three suborders.[65] The suborder Nematocera includes moth flies, midges and gnats, all of which contain members that are

disease vectors and are blood-sucking insects. The suborder Brachycera includes the large-bodied horseflies and deer flies. One genus, Chrysops, blood-sucking flies, includes the vector of tularemia and of loasis, a filarial disease. The remainder are important to man only as pests, many not being blood suckers. The suborder Athericera includes the common housefly and related "filth" flies. They are important in the mechanical transmission of disease, especially under primitive environmental sanitation and on the battlefield; many of their larvae are myiasis producing. Some are vicious biters and pests of man. One genus, Glossina, contains the vector of African sleeping sickness.

Identification of flies depends on classification into suborders on their antennal characteristics and thereafter on the structure of their mouth parts, wings and body. Any dipterous insect possessing mouth parts capable of piercing the skin of man must be regarded as a potential vector of blood-inhabiting, pathogenic micro-organisms; insects with nonpiercing mouth parts obviously cannot be responsible for introducing infection directly into the circulation except through previously injured surfaces. Thus, by inspection of the mouth parts, the insect's disease-conveying hazard may be estimated.

Nematocera. Of the suborder Nematocera, family Psychodidae, only one genus, Phlebotomus, is blood sucking and responsible for transmitting disease.[66] Pappataci fever (sandfly fever),[67, 68] bartonellosis (oroya fever)[69] and leishmaniasis (kala azar and oriental sore[70]) are transmitted by Phlebotomus. In addition to being vectors the sandflies are vicious biters.[71] Phlebotomus flies are minute in size and have extremely hairy bodies and wings. As in mosquitoes, only the female is capable of biting and spreading disease. Eggs of these flies are deposited in batches of fifty in dark, moist crevices in walls, caves and embankments. The larvae feed on organic and excremental debris. After four months a naked pupa is formed, from which the adult emerges; the life cycle is six to eight weeks from egg to adult.

The adults of the nonbiting species of Psychodidae are frequently found in kitchens and outhouses, along creeks filled with decaying matter and about sewage-disposal plants. The larvae are found in decaying vegetable matter, sewage, manure, exuding sap of trees and foul streams.

The Phlebotomus sandflies lay their eggs in crevices of rocks and in damp cracks in shady soil or floors.[72] The adult sandfly varies from 1 to 3 mm. in length and hence can crawl through the meshes of an ordinary mosquito net. Its bite is extremely annoying and may cause a great deal of irritation. The females feed exclusively on blood; they often feed once and die after oviposition, but most species feed at least twice or oftener. They are nocturnal and seldom bite except at night; in some places they seem to forage for only an hour or two after sunset. During the day they hide in darkened corners, cellars and crevices and under floors and rocks. Their flight is limited, and when disturbed on walls, they usually fly only a few inches. Their breeding places are nearly always within a few hundred feet of where they feed. Dimethyl phthalate has been used as a repellent in control of Phlebotomus.[71]

The same suborder contains the family Simulidae, which has only one disease-transmitting genus, *Simulium*, the buffalo gnats and black flies. Species of Simulium act as vectors of larvae of Onchocerca volvulus, the filaria responsible for onchocerciasis.[73] Female buffalo gnats are also vicious and persistent biters. The eggs, in masses of five hundred, are small, glistening and yellow; they are laid on stones and vegetation in swiftly running streams. The larvae, by a posterior sucker, attach themselves to rocks and maintain their position in the current. The brown or green larvae move from one spot to another and tend to congregate in dense, slimy, mosslike groups on stones and other substrates. The full-grown larvae become enclosed in cone-shaped cocoons below the surface of the water. The entire life cycle requires ten to thirteen weeks, but Simulium may hibernate through the winter either in the egg or in the larval stage. Since black flies breed in running water, the methods to be employed in their extermination are quite different from those ordinarily used. The reducing of river flow by damming at intervals, leaving falls between, or, in the case of small brooks, the construction of underground channels are effective measures. In some instances, sweeping stones and other substrates with brooms is of value. Furthermore, the clearing of debris from a stream deprives the larvae of a fastening place. Repellents that are effective against mosquitoes also afford protection against black flies. These flies rarely enter houses, hence fumigation is usually unnecessary. Smudges are beneficial for the protection of both men and animals. The black fly bites freely during daylight.

A third family, Ceratopogodinae, of the same suborder, Nematocera, contains sandflies, no-see-ums, biting midges and punkies. This family includes three genera, of which one, Culicoides,[74] is the vector of filaria. Although species of Acanthocheilonema and Mansonella are considered nonpathogenic to man, they are extremely annoying to both man and animals wherever they are abundant. These flies are extremely small and have light and dark ornamental markings on the wings. Only the female bites. Eggs are usually laid in water or water-saturated sand or soil. The white threadlike larvae develop into slender brown pupae, which may be mistaken for mosquito pupa because of two short breathing tubes on the thorax. The life cycle requires six to twelve months; Culicoides go through the winter either in the egg or in the larval stage.

The great majority of the species that attack man

and animals belong to the genus Culicoides, which lays its eggs on or near water, the larvae living in water or in water-saturated mud or sand. Many favor brackish or salt water. A few species belong to other genera, Leptoconops and Forcipomyia, which breed for the most part in damp places under bark, stones and moss. Nearly all the blood-sucking species become active at dusk; if disturbed, they also bite on bright days in the shade. They are active fliers, often going over half a mile in search of food, but only when the air is still. The bites of Culicoides produce nettle-like pricks, which may be followed by burning sensations and intolerable itching. Control by larvicides is impractical; smoke smudges and oily repellents seem to be more effective. Because of their small size they can penetrate ordinary screen and nets.

Brachycera. The suborder Brachycera contains only one family, Tabanidae, with only one genus, Chrysops, that transmits human disease. Horseflies, deer flies and mango flies are common names for flies of this suborder. Females of the species of the genus Chrysops are vectors of tularemia[40] and loasis.[75] Two other genera, Tabanus and Hematopota, are important only as pests. Some species act as pests to animals and transmit certain animal diseases, such as anthrax, surra, el dehab and swamp fever; they have also been incriminated in Russia as vectors of tularemia.[76] Eggs, in gelatinous masses, are laid on leaves of aquatic plants overhanging sloughs and swamps. In about a week, the larvae hatch and drop into the water or moist ground. They are elongated, tapering sharply at both ends, and have marked ridges at each segment. In several months the fully developed larvae work their way into drier ground, pupate and in two or three weeks emerge as adults. The life cycle requires three to twelve months; overwintering occurs in the egg or larval stage.

Because of their peculiar breeding habits, horseflies, deer flies, mango flies, green-headed flies and other Tabanidae are difficult, if not altogether impossible, to control in the immature stages. Oil films on water may be effective in killing larvae as they hatch from eggs and drop into the underlying water.

Athericera. This suborder, family Musidae, contains one genus, Glossina, that is a vector of a human disease, African sleeping sickness. Other genera of Musidae are important as mechanical carriers of typhoid and paratyphoid fevers, cholera and bacillary and amebic dysentery. In a large number of genera, the larvae may be tissue parasites of man, producing myiasis. The blood-sucking group of these flies include the genera Glossina,[77] Stomoxys and Haematobia; they are vicious biters and vectors of disease. The nonblood-sucking flies include the genera Musca, Fannia, Hippelates, Sarcophaga, Calliphora, Chrysomyia and Lucilia. They are mechanical transmitters of disease and household pests to man.

The genus Glossina, which includes all species of tsetse flies,[78] is chiefly limited to tropical Africa; these flies are the vectors of Gambian and Rhodesian sleeping sickness. Both the male and female are blood sucking. The female is viviparous and carries a single larva at a time. This larva is deposited, fully developed, in dry sandy soil in shady places. The larva burrows down about 5 cm. and pupates. One species occurs in the southwestern corner of Arabia. Tsetse flies[79] are by no means evenly distributed over these great areas, being limited locally to the so-called "fly-belts," chiefly along rivers and at the edges of lakes. They are migratory, especially from minor water collections that may be drying up to larger and more permanent bodies of water. They are diurnal in habits. *Glossina palpalis* is most active in the middle of bright days, and *G. tachinoides* on dull days and in the morning. *G. morsitans* is active in the morning and afternoon but disappears at midday; *G. brevipalpis* and *G. longipennis* bite from sunrise until about 8:00 a.m. and from about 4:00 p.m. until dark. Tsetse flies are attracted by color, especially black and brown, and are repelled by white. When accompanied by Negroes, a white man is seldom troubled by these flies. Black or dark clothes are preferred to light-colored ones, and khaki appears particularly attractive to them. Insect repellents, flyproof clothing or veils and the wearing of white clothing are effective control measures. Passing through the fly-belts at night is the best method of avoiding tsetse flies. Clearing away brush along streams infested with *G. palpalis* and *G. tachinoides* is effective in preventing breeding. Tsetse flies seldom go more than fifty yards from the brushy borders of streams except when following prey, when they may fly several hundred yards. Therefore, if brush is cleared away for thirty yards from the edges of water in the vicinity of fords, villages, washing places and so forth, the flies quickly disappear.

Stomoxys, the stable fly, has been frequently used in the experimental transmission of disease. Although naturally infected specimens have not been found, it is best to consider it as a potential vector of tularemia and other diseases. It is about the size of the common housefly, often enters houses, especially before rains, and is responsible for the belief that the housefly bites.

Stomoxys calcitrans may be noted resting in sunny places on the walls of stables and farm buildings.[80] It breeds primarily in excrement and often can be recovered in abundance in scattered material at the edges of old hay- and strawstacks and in the organic debris often washed up along the seashore. The flies are cosmopolitan in distribution, and control measures are principally local procedures to eliminate breeding in such material.

Species of Haematobia, horn flies, are a serious pest of cattle but rarely attack man. They are blood suckers and closely resemble the housefly.

Musca domestica, the common housefly, develops in animal and vegetable refuse that accumulates about farms and houses.[81] It is almost ubiquitous since its preferred larval foods are manure and fecal wastes, which are usually available near human habitation. It is responsible for the mechanical transfer of enteric disease-producing micro-organisms from fecal material to food.[82-84] It is often mistaken for the stable fly, *Stomoxys calcitrans*, and the latrine fly, *Fannia scalaris*. The latter is frequently seen hovering in mid-air in the middle of the room.

The housefly develops by complete metamorphosis, through the egg, larval, pupal and adult stages each of which requires certain definite environmental conditions.[85] The white, glistening oval eggs are about 1 mm. in length and are deposited in masses of one hundred to one hundred and fifty on or in moist organic matter. The female lays two to four batches in her lifetime. At 85 to 90°F., the eggs hatch in eight hours; at temperatures of 60 to 68°F., in twenty-four hours; and below 40°F., in two or three days. The cylindrical, gray or creamy-white, segmented, wormlike larvae or maggots are highly mobile and burrow into the fermenting mass, reaching maturity in four or five days under favorable conditions. The larvae leave the moist warm environment and migrate to pupate in a dryer and cooler place. The immobile, brown pupal stage lasts three to ten days, whereupon the adult fly emerges, crawls upward through the soil or manure and, as soon as the wings harden, is ready to fly. The female reaches sexual maturity and oviposition begins in from two and a half to twenty days. Under proper environmental conditions the egg-to-adult metamorphosis may be as short as seven days. The optimal temperature for flies is about 90°F.; they are quickly killed in wet material at 108°F. or by a temperature of 115°F. in a dry environment. The housefly does not bite. It cannot ingest particles larger than 0.045 mm. in diameter. Larger substances, such as grains of sugar, are first dissolved by regurgitation of liquid. The housefly is attracted to food principally by odor; it is a persistent and greedy feeder, storing food in its crop, an esophageal diverticulum. The normal flight range of the housefly does not exceed one thousand yards; however, if food is lacking they may migrate as far as a mile, principally in the direction of the prevailing winds. The usual life span is about one month. In the fall many flies are killed by a parasitic fungus disease, *Empusa muscae*. Houseflies are rendered inactive by low temperatures and are killed at 10 to 15°F. They hibernate over winter in the larval or pupal stage.

Green-bottle flies belong to the genera Lucilia and Chrysomyia. They are scavenger flies, depositing their eggs on fish and decaying meat, on human and animal excrement and in open wounds. The thorax and abdomen of Lucilia are metallic green or bluish green and lustrous. The life cycle is similar to that of the housefly. The gray "flesh" flies, Sarcophaga, have a large body. The female is larviparous. The bluebottle flies, Calliphora, are scavengers, feeding on fish, decaying meat and human or animal excrement. They are of robust size and have a blue or metallic sheen to their bodies. All these flies may act as mechanical vectors of enteric infections.

The eye gnats, Hippelates, are vectors of yaws and of infectious conjunctivitis.[86] They are small in size and persistently return to feed on lachrymal secretions even though repeatedly brushed away. They do not bite but have spines on their mouth parts that cause lesions of the skin, through which infection may take place.

Certain species of flies can produce myiasis, a condition wherein fly maggots or larvae live parasitically in the organs and tissues of man and animals. Clinically, myiasis may be classified according to the part of the body invaded.[87] The larvae of *Chrysomyia bezziana* are the usual cause of human myiasis in India. Other examples are species of Dermatobia,[88] tropical warble flies, and of Hypoderma, cattle botflies. Other flies that normally live in decomposing matter but which occasionally lay eggs in living tissues, being attracted by foul discharges and blood, are *Chrysomyia macellaria*,[89] the American screw worm, and species of Lucilia, green-bottle flies and of Sarcophaga, gray flesh flies.

The eggs or larvae of some flies may be swallowed, usually in food or drink. The larvae often develop in the intestine,[90] but this occurrence is accidental and not a part of the normal development. Examples of this type of myiasis-producing flies are included in the genera Calliphora, Sarcophaga, Musca, Fannia, Eristalis and Stomoxys.

(*To be continued*)

References

1. Parran, T. Public health implications of tropical and imported diseases; strategy against global spread of disease. *Am. J. Pub. Health* **34**:1-6, 1944.
2. Fox, L. A. Bacterial warfare: use of biologic agents in warfare. *J. Lab. & Clin. Med.* **28**:539-553, 1943.
3. Jolliffe, N., McLester, J. S., and Sherman, H. C. Prevalence of malnutrition. *J. A. M. A.* **118**:944-950, 1942.
4. Sigerist, H. E. War and medicine. *J. Lab. & Clin. Med.* **28**:531-538, 1943.
5. Councell, C. E. War and infectious disease. *Pub. Health Rep.* **56**: 547-573, 1941.
6. Hackett, L. W. *Malaria in Europe*. 336 pp. London: Oxford University Press, 1937.
7. Coggeshall, L. T. Malaria as world menace. *J. A. M. A.* **122**:8-11, 1943.
8. Meleney, H. E. Problem of certain tropical diseases in war. *New Eng. J. Med.* **227**:159-162, 1942.
9. Manson-Bahr, P. *Synopsis of Tropical Medicine*. 224 pp. London: Cassell & Company, 1945.
10. Medical Department, Pan American Airways, Africa. Development of medical service for airline operations in Africa. *War Med.* **3**:484-497 and 619-634, 1943.
11. Hammon, W. M. Present importance of tropical diseases. *California & West. Med.* **61**:145-149, 1944.
12. Medicine and the war. Tropical diseases in returning military personnel. *J. A. M. A.* **123**:1052, 1943.
13. Draper, W. F. Wartime responsibilities of United States Public Health Service. *New Eng. J. Med.* **229**:639-642, 1943.

14. Roddis, L. H. Organization of Navy Medical Department. *S. Clin. North America* **21**:1533-1543, 1941.
15. Russell, P. F. Military malaria control. *War Med.* **3**:565-584, 1943. Correction. *Ibid.* **4**:224, 1943.
16. Getting, V. A. Tropical diseases in New England. *Science Education.* Vol. 28, 1944.
17. Getting, V. A. Insects and rodents in relation to public health in war time. *J. Pest Control Industry* **2**:8-10, 20-24, 1943.
18. Farner, D. S. Arthropod-borne diseases in Micronesia. *U. S. Nav. M. Bull.* **42**:977-989, 1944.
19. Biraud, Y. Present menace of typhus fever in Europe and means of combating it. *Bull. Health Organ., League of Nations* **10**:1-64, 1943.
20. Dunham, G. C. *Military Preventive Medicine.* Third edition. 1198 pp. Carlisle Barracks: Medical Field Service School, 1938.
21. Simmons, J. S. Preventive medicine program of United States Army. *Am. J. Pub. Health* **33**:931-940, 1943.
22. Mumford, E. P. Mosquitoes, malaria and war in Pacific. *Science* **96**: 191-194, 1942 and *J. Trop. Med.* **45**:74-76, 1942.
23. Shelley, M. D. Winged death. *Hygeia* **21**:636, 1943.
24. Wales, H. G. Q. Malaria and war in Pacific. *Hygeia* **21**:102, 1943.
25. Strong, R. P. *Stitt's Diagnosis, Prevention and Treatment of Tropical Diseases.* 2 vol. Sixth edition. 1747 pp. Philadelphia: The Blakiston Company, 1942.
26. Carson, D. A. Observations on dengue. *U. S. Nav. M. Bull.* **42**: 1081-1084, 1944.
27. Pullen, R. L., and Sundeson, L. Dengue fever. *Am. J. Nursing* **44**: 842-844, 1944.
28. Kisner, P., and Lisansky, E. T. Analysis of epidemic of dengue fever. *Ann. Int. Med.* **20**:41-51, 1944.
29. Dagny, R. H. Aedes scutellaris hebrideus Edwards: probable vector of dengue in New Hebrides. *War Med.* **5**:292, 1944.
30. Simmons, J. S. Global malaria. *New Eng. J. Med.* **229**:605-610, 1943.
31. Bayne-Jones, S. Typhus. *Am. J. Nursing* **44**:821-823, 1943.
32. Endemic typhus fever in 1943. *Bull. U. S. Army M. Dept.* No. 78, 1944. Pp. 24 and 25.
33. Worden, I. K. Scrub typhus fever. *Am. J. Nursing* **44**:942-944, 1944.
34. Huff, C. G. *A Manual of Medical Parasitology.* 88 pp. Chicago: The University of Chicago Press, 1943.
35. Beklemishev, W. N. Comparative study of life-histories of blood-sucking arthropods. *Trop. Dis. Bull.* **40**:511, 1943.
36. Warren, L. H. Military dermatology. *Ann. Int. Med.* **20**:395-404, 1944.
37. Obermayer, M. E. Medical entomology in relation to tropical dermatoses. *J. A. M. A.* **123**:454-458, 1943.
38. Culbertson, J. T. Filariasis. *Am. J. Nursing* **44**:637-639, 1944.
39. Simmons, J. S. *Insects as Vectors of Virus Diseases.* 907 pp. Cambridge: Harvard University Press, 1943.
40. *The Control of Communicable Diseases.* 87 pp. New York City: American Public Health Association, 1943.
41. Froes, H. P. Present knowledge of world problem of yellow fever with special reference to South America and more particularly, to Brazil. *Puerto Rico J. Pub. Health & Trop. Med.* **20**:46-51, 1944.
42. Hammon, W. McD., and Reeves, W. C. Unpublished data.
43. Mitamura, T., et al. Über den Infektionsmodus der japanischen epidemischen Encephalitis mit besonderer Berücksichtigung der experimentellen und epidemiologischen Beweise für die Krankheitsübertragung durch Mücken. *Tr. Soc. path. jap.* **28**:135-145, 1938.
44. Hammon, W. McD., and Reeves, W. C. Laboratory transmission of St. Louis encephalitis virus by three genera of mosquitoes. *J. Exper. Med.* **78**:241-253, 1943.
45. Russell, P. F. War and public health. *New Eng. J. Med.* **231**:255-259, 1944.
46. Soper, F. L., and Wilson, D. B. *Anopheles Gambiae in Brazil.* 262 pp. New York City: The Rockefeller Foundation, 1930-1940.
47. Dyar, H. G. *The Mosquitoes of the Americas.* 621 pp. Washington, D. C.: Carnegie Institution of Washington, 1928.
48. *Idem. The Mosquitoes of the United States.* Proceedings of the United States Natural Museum. 119 pp. Vol. 62, 1922.
49. Johannsen, O. A. *Aquatic Diptera.* Part I. *Nemocera, exclusive of Chironomidae and Ceratopogonidae.* Ithaca, New York: Cornell University Agricultural Experiment Station Memoir 164. June, 1934.
50. Matheson, R. *Handbook of the Mosquitoes of North America.* Springfield, Illinois: Charles C Thomas, 1929.
51. Feemster, R. F., and Getting, V. A. *Special Report of the Department of Public Health Relative to Varieties and Prevalence of Mosquitoes in the Commonwealth.* Report to Massachusetts Legislature, House No. 2260. 70 pp. December, 1940.
52. Getting, V. A. Tropical disease and global war. *Am. J. M. Sc.* **207**: 404-413, 1944.
53. Johnson, C. W. Fauna of New England: 15th list of diptera or two-winged flies. Occ. Papers, Boston Soc. Nat. Hist. VII, Boston, February, 1925.
54. Brues, C. T., and Melander, A. L. *Key to the Families of North American Insects.* Boston and New York City: Privately printed, February, 1929.
55. Tulloch, G. S. *Key to Mosquitoes of Massachusetts: Psyche.* Vol. 46, 1939. Pp. 113-136.
56. Komp, W. H. *A Guide to the Identification of the Common Mosquitoes of the Southeastern United States.* Reprint No. 636. *Pub. Health Rep.* May, 1923. Revised, 1936.
57. *Massachusetts Mosquito Survey: Training course for field personnel.* 90 pp. Edited by V. A. Getting. Massachusetts Department of Public Health, 1939.
58. Howard, L. O., and Bishopp, F. C. Mosquito remedies and preventives. *Farmers' Bull., U. S. Dept. of Agriculture.* No. 1570. June, 1932.
59. Hollis, M. D. Modern malaria control. *Am. J. Pub. Health* **34**:494-498, 1944.
60. Mackie, T. T. Malaria in army. *Ann. Int. Med.* **20**:655-660, 1944.
61. McCoy, O. R. Malaria and war. *Science* **100**:535-539, 1944.
62. Hargett, M. V. Control of yellow fever. *Puerto Rico J. Pub. Health & Trop. Med.* **20**:3-24, 1944.
63. Cochrane, E., and Newbold, C. E. Notes on design and performance of flushing siphon. *Ann. Trop. Med.* **37**:108-114, 1943.
64. Butler, F. A. Malaria control program on South Pacific base. *U. S. Nav. M. Bull.* **41**:1603-1612, 1943.
65. Davidson, J. Flies, fleas and lice. *M. J. Australia* **2**:111-116, 1942.
66. Cullinan, E. R., and Whittaker, S. R. F. Outbreak of sandfly fever in two general hospitals in Middle East. *Brit. M. J.* **2**:543-545, 1943.
67. Sabin, A. B., Philip, C. B., and Paul, J. R. Phlebotomus (pappataci or sandfly) fever. *J. A. M. A.* **125**:603-606, 1944.
68. Sandfly fever. *Bull. U. S. Army M. Dep.* No. 79, 1944. P. 1.
69. Fox, H. Tropical diseases of skin. *New Eng. J. Med.* **231**:482-485, 1944.
70. Ball, D., and Ryan, R. C. Cutaneous leishmaniasis. *Bull. U. S. Army M. Dept.* No. 79, 1944. Pp. 65-73.
71. Philip, C. B., Paul, J. R., and Sabin, A. B. Dimethyl phthalate as repellent in control of phlebotomus (pappataci or sandfly) fever. *War Med.* **6**:27-33, 1944.
72. Fox, H. Yaws, cutaneous leishmaniasis and pinta. *J. A. M. A.* **123**: 459-462, 1943.
73. Blacklock, D. B. Development of Onchocerca volvulus in Simulium damnosum. *Ann. Trop. Med.* **20**:1-48, 1926.
74. Fox, I., and Hoffman, W. A. New neotropical biting sandflies of genus Culicoides (Diptera: Ceratopogonidae). *Puerto Rico J. Pub. Health & Trop. Med.* **20**:108-111, 1944.
75. Guy, W. H., Cohen, M., and Jacob, F. M. Infection with Loa loa. *Arch. Dermat. & Syph.* **47**:763-767, 1943.
76. Olsufiev, N. G. Nouvelles donnees experimentales sur la transmission de l'infection tularemique par les taons (Tabanus). *Med. Parasit. and Parasitic Dis.*, Moscow, Vol. 9, No. 3, 1940.
77. Ferreira, C. Trypanosomiasis — Alguns aspectos da tripanosomiase humana no noroeste da Colonia de Mocambique. *Bol. Geral de Med. Bastora.* Ser. 24. Nos. 7-9, 1942. Pp. 100-109.
78. Duke, Kleine, Lavier, Prates, Peruzzi and Van Hood. *Final Report of League of Nations International Commission on Human Trypanosomiasis.* Geneva, 1938.
79. Swynnerton, C. F. M. Tsetse flies of East Africa. *Trans. Roy. Entomolog. Soc. London* **84**:36 and 576, 1936.
80. Rosenau, M. J. *Preventive Medicine and Hygiene.* Sixth edition. 1491 pp. Boston: MacMillan, 1940.
81. Strong, R. P.[28] P. 1732.
82. Gwatkin, R., and Mitchell, C. A. Transmission of *Salmonella pullorum* by flies. *Canad. Pub. Health J.* **35**:281-285, 1944.
83. Kuhns, D. M., and Anderson, T. G. Fly-borne bacillary dysentery epidemic in large military organization. *Am. J. Pub. Health* **34**: 750-755, 1944.
84. Baer, L. S., and Allen, R. F. Prevention of fly-borne diseases in islet and atoll warfare. *Mil. Surg.* **94**:296-301, 1944.
85. Howard, L. O., and Bishopp, F. C. House fly and how to suppress it. *Farmers' Bull. U. S. Dept. of Agriculture,* No. 1408, 1926.
86. Kumm, H. W. *Report of Jamaica Yaws Commission for 1934.* Vol. 19, 1935.
87. Basso, R. Myiasis in Mendoza, Argentina. Univ. Buenos Aires: Mision de Estudios de Patalogia Regional Argentina. No. 41, 1939. P. 55.
88. Harrell, W. B., and Moseley, V. Surgical treatment of subdermal myiasis due to Dermatobia hominis. *South. M. J.* **35**:720-723, 1942.
89. Cushing, E. C., and Patton, W. S. Studies on higher Diptera of medical and veterinary importance: Cochliomyia americana sp. nov., screw-worm fly of New World. *Ann. Trop. Med.* **27**:539-551, 1933.
90. Causey, O. R. Experimental intestinal myiasis. *Am. J. Hyg.* **28**: 481-486, 1938.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 31111

PRESENTATION OF CASE

A forty-three-year-old factory proprietor was admitted to the hospital because of increasing dyspnea and dependent edema.

The patient was well until three years prior to admission, when he developed shortness of breath on exertion, edema of the ankles and legs, and weakness. He was admitted to a community hospital, where he was treated for heart failure over a period of four weeks, following which he returned to work believing himself to be entirely well. He continued to work and, except for an increasingly bloated feeling, was essentially free from symptoms until about one year prior to entry, when he again developed dyspnea and edema, progressing to a point that made hospitalization necessary. He remained in the hospital for twelve weeks and was discharged improved, but some dyspnea and edema were still present. He was unable to return to work, remaining at home resting and receiving diuretic injections once or twice a week. In spite of these measures, he noted a gradually increasing bloated feeling and abdominal distention. He was admitted to the hospital for study.

The patient recalled no childhood illness or definite rheumatic infection. He had had an attack of gonorrhea thirteen years previously and had recovered promptly after treatment. He had lost about 20 pounds in one and a half years.

Physical examination revealed a rather thin man in no acute distress who became somewhat short of breath on slight exertion. The skin appeared moderately atrophic over the whole body, and there was little subcutaneous tissue. Pitting edema was present, most marked over the sacrum, extending up the back, and to some extent in the lower extremities. There was questionable early clubbing of the fingers. The pupils reacted slowly to light and accommodation. There was some stiffness and limitation of motion of the neck in all directions, with poor muscles and a large, dilated, pulsating vein on the right. There was moderate scoliosis, with the thoracic concavity to the right, and slight lordosis. The thoracic cage showed little motion and flared out asymmetrically. There was dullness at both bases, more marked on the left, with decreased fremitus and diminished breath sounds. No rales were heard. The heart appeared enlarged. The left border of dullness was about 9.5 cm. from the midsternal line and 1 cm. outside the midclavicular line. There was questionable enlargement to the right. The point of maximum impulse could not be felt. The rhythm was irregular, with an extra beat and an occasional coupling for short periods. The sounds were of fair quality. A Grade 2 to 3 systolic murmur was audible over the lower end of the sternum and apex. No diastolic murmurs were heard. The liver extended to the level of the umbilicus. It was firm and nontender, with a smooth surface and a smooth sharp edge. The spleen was easily palpable, firm and nontender. The radial arteries were thickened but not tortuous, and no pulsations could be felt in the pedal vessels. There was some limitation of motion in all joints, especially the elbows, but there was no pain or crepitus on manipulation.

The temperature was 98.6°F., the pulse 50, and the respirations 16. The blood pressure was 110 systolic, 80 diastolic.

Examination of the blood showed a red-cell count of 4,610,000, with 13.6 gm. of hemoglobin, and a white-cell count of 10,200, with 78 per cent neutrophils, 8 per cent lymphocytes, 10 per cent monocytes, 2 per cent eosinophils and 2 per cent basophils. The red cells and platelets were normal. The urine had a specific gravity of 1.016, with a + test for albumin and a few white cells in the sediment. The serum protein was 4.4 gm. per 100 cc. A blood culture was negative. A blood Hinton reaction was negative. A bromsulfalein test showed 15 per cent dye retention in the serum. An antecubital-vein-to-mouth circulation time with Decholin was 36 seconds. The venous pressure ranged between 12 and 15 cm., the veins filling and emptying with the heart cycle.

A roentgenogram of the chest revealed considerable fluid in the left pleural cavity obscuring the diaphragm and producing some compression of the lower lobe. A small amount of fluid was also present in the right costophrenic sinus. The visible lung fields were clear except for a healed Ghon's complex on the right, with calcification in both hili. The heart showed a marked general enlargement, with a transverse diameter of 17 cm., as compared to an internal chest diameter of 27.5 cm. Pulsations of the heart were slow, but the amplitude was within normal limits. There was no dilatation of the left auricle. Examination of the esophagus showed no delay in the passage of barium and no evidence of varices.

An electrocardiogram on admission (Fig. 1) revealed an almost regular ventricular rhythm, with a rate of 60, and frequent ventricular premature beats from varying foci. There was no discernible auricular activity. There was an extremely low

*On leave of absence.

voltage of the QRS complexes, and almost flat T waves in the standard leads, with slight sagging of ST_1 and ST_3. Lead 4 showed an absent R, a slightly elevated ST and a flat T.

The patient was placed on a cardiac regimen, including a low-salt diet, limitation of fluids, 0.1 gm. of digitalis daily, ammonium chloride and Mercupurin. An electrocardiogram on the seventh hospital day showed no change except for disappearance of the ventricular premature beats and a somewhat slower rate. Precordial leads revealed a tendency to low voltage, with absent R waves in Leads 1, 2, 3 and 4. The T waves were low and upright in Leads 1 and 2, diphasic in Lead 3 and inverted in Leads 4, 5 and 6.

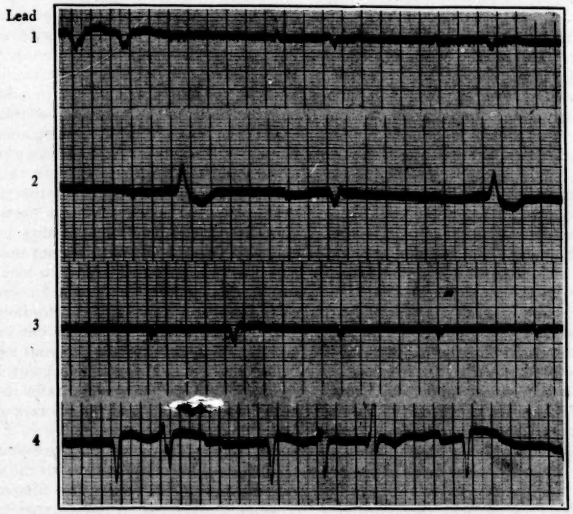

FIGURE 1. *Electrocardiogram on Admission.*

The patient continued unchanged except for a brief upper respiratory infection on the tenth and eleventh hospital days, during which the temperature rose to 102°F. but rapidly returned to normal. The pulse continued slow. A left thoracentesis on the twenty-third hospital day was productive of 150 to 200 cc. of clear straw-colored fluid, which was negative on culture.

On the twenty-fourth hospital day an operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. C. SIDNEY BURWELL*: I should like to draw the picture of the way this disease began. In the first place, it seems to me significant that the patient at the time of onset was forty-three years old — not twenty-three, or sixty-three, but forty-three. In the second place, he had been having symptoms for something in the neighborhood of three years; for over a year these had been more or less fixed but present most of the time. It seemed to me as I read the story that over a three-year period he had had rather less fluctuation in the course of his disability than many people with progressive types of cardiac disease. I began to think about the kinds of cardiac disease, if this should turn out to be cardiac disease, which can give a slowly progressive, fixed type of disability. There are several conditions that will do that.

One immediately thinks of tricuspid stenosis. With tricuspid stenosis there is apt to be a long history of peripheral congestion, with enlargement of the liver, often with edema but without the fluctuations that are frequent in people who are suffering from variations in the capacity of the myocardium. There are many reasons to believe that this man did not have tricuspid stenosis. In the first place, he did not have mitral stenosis, and to have tricuspid stenosis without its older brother is extremely unusual. He had no signs of it in his murmurs, and no suggestion in his x-ray films, nor did he have the degree of venous distention that is usually compatible with that as a primary situation.

If he did not have tricuspid stenosis, had he some other variety of obstruction? It is striking that he had no evidence of acute congestion of the lungs,

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that he had primarily peripheral congestion and that the dyspnea was never paroxysmal and was not present at rest but occurred when he tried to exert himself. This suggests that the dyspnea was due to inability on his part to increase cardiac output or the respiratory volume.

Another type of obstruction to circulation that he might have had is pericardial disease. If he had pericardial disease with this history it would certainly have been chronic pericardial disease, and it might have been that interesting disorder called "constrictive pericarditis," which Dr. White[1,2] began to study actively in 1928. I shall come back to that presently.

A third possibility is pulmonary heart disease with cor pulmonale. This would account for slow progression without much fluctuation. Cor pulmonale is an interesting disorder, which may be based on a good many kinds of changes in the pulmonary circulation and which is usually marked by right ventricular failure. Perhaps he had right ventricular failure, but he had no cyanosis, no evidence of extensive lung disease, no history of cough anywhere in this record. If he had cor pulmonale he must have had an unusual form of primary pulmonary hypertension, and I do not find any evidence of it. There are other disorders that lead to right-sided failure, and the most frequent is mitral stenosis. He might have had mitral stenosis, with right ventricular failure of a rather fixed type, but the evidence in the murmurs, in the history and in the x-ray films is against it.

Could this form of symptoms result from a myocardial infarct on the basis of vascular disease? Occasionally, although rarely, one sees vascular degeneration of the myocardium almost limited to the right ventricle. In such a situation this history might be possible.

I want to consider in a moment the possibility that this was not cardiac disease at all, at least not primarily, because there are certain interesting suggestions that other organs were involved in an important way. Before doing that, I should like to review and perhaps get a little help on one or two bits of evidence that are presented. In the first place, the venous pressure is recorded as equivalent to 12 to 15 cm. of water. It is particularly necessary in a man with an abnormal chest to know how that was measured. How was the zero point determined?

DR. EDWIN O. WHEELER: It was determined by measuring 5 cm. down from the sternum rather than 10 cm. up from the back.

DR. BURWELL: I should also like to know what the anteroposterior diameter of this man's chest was.

DR. WHEELER: I do not know.

DR. BURWELL: Could you guess? Was it increased or normal?

DR. PAUL D. WHITE: I should say that it was normal.

DR. BURWELL: That is comforting. On this x-ray film it looks increased. Would you agree, Dr. Holmes?

DR. GEORGE W. HOLMES: Yes.

DR. BURWELL: These measurements are important. Assuming that this is a cross section of the chest and the heart is lying here, if you measure 5 cm. down from the sternum, you hit the level of the right auricle. If you measure 10 cm. up from the back, you hit about the same level. If a man has an emphysematous chest, that relation of the heart to the back does not change, but the anterior chest wall in effect is lifted off the heart. Thus, if you measure 5 cm. back from the sternum, you set your level above the right auricle and therefore get a false low venous pressure. Every now and then one sees obvious right ventricular failure and a normal venous pressure, and in a fair percentage of these patients the normality of the venous pressure is the result of error in the placing of the zero point. When you put it somewhere near the position of the right auricle you find that the patient has a high pressure. I take it that this man had venous distention and a venous pressure that was above the usual level of normality by the Moritz and von Tabora[3] method, but not much. I am inclined to regard 12 to 15 cm. as a high venous pressure in this man. He had a thick chest, so that one should probably add 5 to 6 cm. to the measurement.

DR. WHITE: What would you put as the top normal measurement according to your method?

DR. BURWELL: Using a zero point 10 cm. above the back, all our measurements in normal persons were under 15 cm., and 90 per cent of them were under 13 cm.

DR. WHITE: These readings are doubtless higher than those one would obtain with the subject in the vertical position. Twelve to fifteen centimeters is actually greater than the distance from the level of the clavicle to where the superior vena cava enters the right auricle; so I think that your readings may be too high from the standpoint of the actual or true venous pressures.

DR. BURWELL: Yes, but the arm-vein pressure is sensibly higher than the auricular pressure. If one takes 100 normal people and measures the venous pressures with the Moritz and von Tabora method, with the zero point 5 cm. down from the sternum, and charts them against the anteroposterior diameters of the chest, one gets, in general, a good correlation. The bigger the chest, the lower the venous pressure. If one charts the venous pressures determined by using the zero point 10 cm. from the back against the diameters of the chest, there is no correlation. It is an important point.

I am satisfied that this man had a high venous pressure. That is what I wanted to be sure of.

DR. WHITE: The neck veins were engorged.

DR. BURWELL: It is true that there is a statement about distended, pulsating neck veins that col-

lapsed and filled with the action of the heart, but it does not say what position the man was in when that was observed and I wanted a little reassurance. I think the question of venous pressure is an all-important one.

There is an interesting variety of cardiac disorder, associated with severe kyphoscoliosis, that is called "pulmonocardiac failure." This was admirably described by Chapman, Dill and Graybiel[4] a few years ago. The disability is largely one of ventilation, and the involvement of the heart in the composition of that picture is relatively small. Here is a man with slight scoliosis, and he may have had some limitation of excursion because of the relative immobility of the chest, but I do not believe that he had heart failure on the basis of the chest deformity, nor do I believe that all the symptoms can be explained on the basis of emphysema, some fixation of the ribs and loss of elasticity of the lungs, although all of these were probably present.

In considering some of the matters that have been tossed up with regard to the varieties of heart disease leading to obstruction or right ventricular failure, two important observations are that the heart was considerably enlarged and that the enlargement was not associated with a great reduction in excursion. An interesting question immediately comes to mind. In the x-ray and fluoroscopic examinations the amplitude of pulsation is said to have been within normal limits. The amplitude of the heart excursion is most closely related to the amount of blood that it expels per beat, and the amount of blood expelled with each beat has an important relation to the rate of the heart. He maintained a cardiac output of 4 liters per minute. The heart does not have to expel as much per beat if beating at a rate of 100 as it would if it were beating at a rate of 50, and the excursion would be smaller at a rate of 100. I should like to know if this excursion was normal for a rate of 50 or normal for a rate of 80.

DR. HOLMES: You have raised an interesting point, and one that I cannot answer. I do not believe that radiologists, at least those in this hospital, pay as much attention to that as they might. When the radiologist observes the heart with the possibility of an adherent pericardium in mind, he notes whether or not the amplitude of the beat is diminished, regardless of the pulse rate.

This heart is somewhat triangular in shape, which is slightly in favor of an adherent pericardium.

DR. BURWELL: The landmarks are not striking. It is rather an expressionless kind of heart silhouette.

DR. HOLMES: There is another point, if one can be certain about it; namely, one cannot distinguish the auricle from the ventricle along the left border. This is a difficult observation, particularly in a patient with fluid in the chest. It is not well seen. With an adherent pericardium or pericarditis with effusion, there is usually widening of the mediastinal shadow above the heart. This man certainly has no widening in that area, but this film was taken to bring out the right border.

The shadow in the lower part of the left chest is obviously fluid. In the films taken sixteen days later, one can see that the fluid has largely disappeared, although not completely.

DR. BURWELL: Do you see any evidence of calcification in the region of the heart?

DR. HOLMES: No. As you pointed out, I do not believe that the patient had enough scoliosis or emphysema to account for any of these things. I suppose that these calcified lymph nodes around the root of the lung are of some interest if one is thinking of tuberculous pericarditis. There is evidence that he had had a tuberculous infection at some time.

DR. BURWELL: The lungs are singularly clear.

DR. HOLMES: Yes, with the exception of this fluid. The interlobar fissure is visible, but I do not believe that it means anything. It may mean that the films were taken at exactly the right tilt.

DR. BURWELL: That is helpful to me.

Dr. White,[5] with his characteristic precision, has recently studied the size of the heart in constrictive pericarditis. Many workers were entranced with the thought that here was a situation where one could observe severe evidence of cardiac failure with a normal-sized heart, and placed considerable emphasis on this point. But being a systematic kind of fellow, Dr. White set out to see how many of his patients with constrictive pericarditis had an enlarged heart, and it came out that 40 per cent had appreciable enlargement. Therefore, the fact that this heart was enlarged does not rule out constrictive pericarditis. But enlargement of this degree is unusual in constrictive pericarditis, not many of anybody's cases having had this much. Therefore one again gets a warning signal.

In favor of constrictive pericarditis is the fact that there is no other obvious explanation for this man's heart failure, except one to which I shall refer in a moment. He had no evidence of valvular disease, no evidence or history of hypertension and no evidence of congenital heart disease that I can find, and in so far as the evidence of these things is missing he falls into the type of persons who might have constriction. He had one other suggestive thing, namely, thick radial arteries. He had no pulsations in his feet and he had a complex electrocardiogram, and the question arises whether this man has to be classified on the basis of vascular degeneration of the myocardium. I allowed the pleasant thought of coarctation to pass through my mind when I read about the feet, but he had no hypertension or other things that go with coarctation and I see no great notches in his ribs, no collaterals and so forth, so that we can lay coarctation aside.

Could he have had some rare kind of occlusive arterial disease that shut off the arteries of the feet

without giving symptoms of interference with the arteries in the myocardium and without causing local symptoms? I think that is unlikely.

I want to talk briefly about the confusing electrocardiograms. There are certain things that are entirely compatible with chronic pericardial disease. The characteristic things in constrictive pericarditis are low voltage and flat T waves. He had both of these, but he had some other things that are suggestive of anterior infarction. There was a slightly elevated ST, and an absent R in the precordial leads, with a variation in T waves in the precordial leads, which is also compatible with anterior infarction. The T waves in the limb leads are not like those of anterior infarction, and the low voltage is much more like pericardial disease than coronary disease in its unanimity in all the leads. I am not at all sure that a good thick layer of something over the myocardium would not interfere with the electrical activity in much the same way that an area of dead muscle in the same position would. I do not see any theoretical reason why a pericardial scar would not imitate an anterior infarction from that point of view. Also, as you well know, one of the characteristics about tuberculous pericarditis is that it is always accompanied by subpericardial myocarditis of varying degree and extent, and this may also lead to abnormal electrocardiographic manifestations. I am therefore inclined to find a good many difficulties in pinning a diagnosis of active coronary disease or of significant coronary occlusion on this man. There was no pain to suggest it, and not the fluctuation in the course that is usually produced by successive occlusions, and I think that the electrocardiogram can perhaps be explained on another basis.

I should like to introduce a discordant note and suggest that perhaps the whole picture was caused by cirrhosis of the liver. He had a number of things that go with cirrhosis of the liver. He had a big liver, a low protein and atrophic skin. On the other hand, he had no ascites, no varices and no spider telangiectases. He had had this thing too long, I believe, without ascites for it to have been cirrhosis of important degree. Moreover, although he had an enlarged spleen, I think that this and the increase in connective tissue in the liver were probably secondary to cardiac congestion and not to a primary cirrhotic process. The hypoproteinemia is rather important in the business of producing his symptoms, because when one has a total protein of 4.4 gm. the chances are that the albumin is severely reduced. The osmotic pressure is correspondingly reduced, and the transference of fluid to tissues is greatly facilitated. Under these circumstances a relative, slight increase in venous pressure may be important in causing edema, and I suspect that, so far as the edema goes, this man's low protein was quite as contributory as the dynamic situation created by his increased venous pressure.

I shall therefore make a diagnosis of constrictive pericarditis in this man, in spite of several points against it and in spite of the fact that not one of the usual cardinal signs was present. He had no evidence of calcium in the pericardium, he had no pulsus paradoxus, he had no ascites, although he had had congestion for three years, and his heart was certainly on the big side. I think more of the findings can be explained by constrictive pericarditis than by anything else. My second choice is localized arterial disease of the right ventricle, with myocardial degeneration, but I think that is a poor second, and I shall rest the case on constrictive pericarditis. If it was that, it was probably due to tuberculosis.

DR. WHITE: Dr. Burwell saw this patient during life but doubtless does not remember him. The note that he wrote at that time is certainly consistent with what he has just said. In December, 1943, he wrote:

> This man has had fixed congestion for about one and a half years, preceded by intermittent congestion for a year. He exhibits distended veins, a feeble apex beat and a small pulse pressure (105/80) in spite of a rate of 48. He has no evidence of valvular disease. He has a large liver and edema. The lung descends in front of the heart. The chest moves in a limited way (vital capacity, 1500 cc.), and there is fluid and probably fibrous pleuritis at the left base. I believe that he has a constrictive pericarditis and that operation is justified. It is possible for extensive myocardial disease to produce the same picture, but I see no evidence of it. Two problems must be considered in the postoperative course — the low protein in the blood and the limited vital capacity. Removal of the pleural fluid would help the latter. Fluoroscopically the impressive things are a moderate increase in heart size and a marked limitation in the movement (excursion) of the heart in spite of the slow rate.

We both examined the patient fluoroscopically, if you recall.

DR. BURWELL: I begin to remember him, but I do not recall what fluoroscopy showed.

DR. WHITE: It seemed to us that he had a large heart that was somewhat limited in excursion. I asked you to come down because I wanted moral support for surgical exploration on account of the possibility of constrictive pericarditis. As a matter of fact, I do not remember exactly what I said myself at that time. Is there a note by me?

DR. WHEELER: We agreed essentially with Dr. Burwell that the case was not characteristic of constrictive pericarditis but that no other good diagnosis could be made. We thought that the man deserved a chance of help by operation.

CLINICAL DIAGNOSIS

Constrictive pericarditis (? type).
Heart disease of other nature?

DR. BURWELL'S DIAGNOSIS

Constrictive pericarditis.

ANATOMICAL DIAGNOSIS

No evidence of constrictive pericarditis.

Pathological Discussion

Dr. Benjamin Castleman: An exploratory thoracotomy, performed by Dr. Sweet on the twenty-fourth hospital day, revealed 100 cc. of clear, straw-colored fluid in the pericardial sac. The sac was not thickened, and no adhesions were present. The pericardial fluid proved to be negative for tubercle bacilli on guinea-pig inoculation.

Following the operation the patient continued as before, with periods of congestion relieved periodically by diuretics, and he was discharged on the forty-first hospital day.

We shall now continue with this patient's second admission, Dr. Burwell, and give you another chance.

Dr. Burwell: That is wonderful. Opportunity is supposed to knock but once.

Additional History

Second admission (six months later). Following discharge the patient seemed somewhat improved and remained up and around on a regimen of limited fluids, digitalis, ammonium chloride and periodic courses of mercurial diuretics. He complained of occasional mild pains and aches in the right shoulder, elbow and fingers, apparently unassociated with fever. He also noted an aching pain, occasionally sharp, over the region of the precordial incision, without radiation or relation to exertion; it did not respond well to morphine. About six weeks prior to admission he became rather suddenly worse, with increased congestion in spite of diuretics; he was confined to bed, and several chest taps were performed. His appetite became poor, and he lost considerable weight. One week before admission he suddenly developed a severe and persistent headache involving the right side of the head, with blurring of vision in the right eye and some weakness of the extremities on the left.

A further history revealed that a younger brother had had symptoms similar to those of the patient, including moderate enlargement of the heart, with auricular fibrillation, and hepatosplenomegaly.

Physical examination showed a wasted man, with a large protuberant abdomen. There was no evidence of facial asymmetry. The neck veins were distended and pulsating. The heart and lung findings were essentially unchanged. The liver was felt five to six fingerbreadths below the costal margin, and there was questionable pulsation. The spleen was easily palpable. Marked pitting edema of the lower extremities was present, more so on the left. The left arm and leg were weaker than those on the right, the left abdominal reflexes were absent, and there were positive Babinski and Chaddock signs on the left.

The temperature was normal, and the pulse continued to range about 60. The blood pressure was unchanged.

Examinations of the blood and urine showed no significant change. A roentgenogram of the chest showed increase in the pleural fluid bilaterally. There were some areas of atelectasis in the right lower lobe, and the left lower lobe appeared somewhat reduced in size. The elbows and dorsolumbar spine showed no abnormalities except for slight decalcification of the latter. An electrocardiogram showed irregularity in the rhythm, with occasional ventricular premature beats, but no other significant change.

Examination of the fundi revealed retinal edema of the right eye that extended downward and nasalward from the disk, with a corresponding defect in the visual field.

Several chest taps were performed to relieve the patient's distress. A lumbar puncture on the ninth hospital day revealed clear colorless fluid under an initial pressure equivalent to 290 mm. of water. No cells were present, and the Wassermann reaction was negative. A muscle biopsy was negative.

On the twentieth hospital day the patient complained of increased difficulty in breathing and of pain in the left chest. Two days later he became nauseated, vomited, complained of vague lower abdominal pain and hiccoughed frequently. On the following morning, the radial pulse was imperceptible and the blood pressure unobtainable. The heart sounds could not be heard. The lungs were full of bubbling rales, especially on the right. The extremities were cold and clammy. The patient rapidly became unresponsive and, in spite of resuscitative measures, died on the twenty-third hospital day.

Further Discussion

Dr. Burwell: An initial spinal-fluid pressure of 290 mm. of water is not significant of what is going on in the central nervous system unless we know the venous pressure, because the former rises precisely with the latter.

This man had three things: a continuation of heart failure, pain in the right shoulder, elbow and fingers and over the precordial incision and some sort of intracranial accident that involved the motor tract on the right and caused a defect in the visual field of the right eye. The only way I can put these together as an alternative to the pericarditis that he did not have is in terms of vascular disease with thrombosis. He had a brother who had had similar symptoms. It was certainly not Wilson's disease. I do not find any way of explaining the cardiac situation on a neoplastic basis; we are fairly sure that he did not have a neoplasm of the pericardium, and there is no reason to think that he had an intracardiac neoplasm. Intracardiac neoplasm tends to be more progressive than this. I am inclined to believe that both the heart failure and the later symptoms were vascular in origin.

Dr. Castleman: By that do you mean infarction of the myocardium?

DR. BURWELL: I mean old infarction of the myocardium, probably multiple ones, with a degenerated myocardium based on cardiac disease (myomalacia).

DR. HOLMES: It may be of help to look at the later films. The heart has not changed in size or shape during the interval, but the patient has developed fluid in the right pleural space as well as in the left. The pleural fluid is persistent.

DR. BURWELL: It certainly is persistent. There is no evidence of an infectious process that would satisfy me. I find it difficult to diagnose tuberculosis of the pleura. The patient had had fluid in the pleura for many years, and that is not the way an active tuberculous lesion of the pleura is apt to do. The possibility of neoplasm in any queer situation is always good. He had a low serum protein, which means that fluid would pour out into the pleural cavity with great ease from slight inflammation or from slight elevation of the venous pressure. I am trying to think of a good alternative in terms of a noncardiac situation in his pleura, but he had so much evidence of injury to the heart in the electrocardiogram and in the size of the heart that I am inclined to believe that the edema, the pleural effusion and the dyspnea on exertion were cardiovascular in origin. I do not believe that he had beriberi, and there is no reason to believe that he had an aortic dissection. I doubt that the queer kinds of heart disease, such as scleroderma, play any role here. I am afraid I cannot do better.

DR. J. H. MEANS: Could he have had a process, not constrictive pericarditis, that constricted the inferior vena cava?

DR. BURWELL: That is an attractive suggestion, but such cases are extremely rare. This man had a high venous pressure in the arms; he should therefore have had something wrong with his heart rather than a localized difficulty.

DR. WHITE: Yes; isolated constriction of the inferior vena cava would protect the heart and prevent an increase in the venous pressure in the arms and neck. It would give a big liver, of course.

DR. MEANS: Was there a mediastinal process?

DR. BURWELL: That is what I was thinking of, an intracardiac tumor where the cava enters, thus plugging up the auricles. I should think that the heart would be smaller, underworked rather than overworked.

DR. WHITE: May I summarize the situation as I recall it? We had Dr. Burwell come down here to see this man because we were frankly puzzled. The patient did not run true to form respecting chronic constrictive pericarditis, but we thought it only right to explore and Dr. Burwell agreed.

After the operation I entered the following note:

> This patient's condition in all its details is unique in our experience, although it resembles somewhat that of one other patient, in that right ventricular enlargement, dilatation and failure are present, unexplained by history, symptoms, signs, laboratory data or even operation. Whether myocarditis, toxic or infectious, or a bizarre coronary heart disease involving primarily the auricles and right ventricle is the etiologic factor we have no way of determining. Certainly the treatment should consist of tonic therapy for the heart and diuretic therapy for the congestion. It is conceivable that with time and the subsidence, spontaneous or otherwise, of the toxic or infectious factor, the right ventricle may regain its tone, but it probably will not do so.

When the patient came to us at the time of the last admission we still had no answer concerning the underlying etiologic factor in his case. I wrote:

> It is possible that a constitutional disease, as suggested by Dr. William Beckman, — for example, dermatomyositis involving the myocardium as well as the other muscles of the body, — may be the answer, but it is simply conjectural at present. I favor further studies along this line. The patient feels a little better.

It was because of Dr. Beckman's suggestion that a muscle biopsy was done. This gave negative results.

DR. BURWELL: Constitutional disease is a good idea, but I should be inclined to think that a man of forty-three who had thick vessels and absent pulsations in the feet, with subsequent hemiplegia, would be likelier to have a vascular disorder, either a precocious form of arteriosclerosis or polyarteritis. The story certainly does not sound like polyarteritis, although that is possible.

DR. WHITE: Do you think that the cerebral lesion might have been the result of embolism?

DR. BURWELL: Yes, it could have been; but I find little evidence for subacute endocarditis. If he had an embolus, it must have come from a mural thrombus.

DR. MEANS: Does periarteritis nodosa produce anything like this?

DR. CASTLEMAN: It can mimic anything.

DR. BURWELL: There is one other point regarding constitutional disease. The patient had a reasonably low white-cell count with a high percentage of monocytes, and perhaps one ought to think of some more active form of tuberculosis. But I do not see any other good evidence of active tuberculosis and am inclined to think that all his difficulties were on a vascular basis. I do not believe that he had syphilis.

CLINICAL DIAGNOSES

Cardiac hypertrophy, cause unknown.
Congestive failure.
Acute pulmonary edema.

DR. BURWELL'S DIAGNOSIS

Myocardial degeneration based on vascular disease.

ANATOMICAL DIAGNOSIS

Interstitial myocarditis.

PATHOLOGICAL DISCUSSION

DR. CASTLEMAN: The autopsy showed a terminal bronchopneumonia and rather severe edema of the

lungs; there was also some fluid in both pleural cavities. The heart was enlarged but not markedly so; this was due mostly to dilatation rather than to hypertrophy. Since the heart weighed only 340

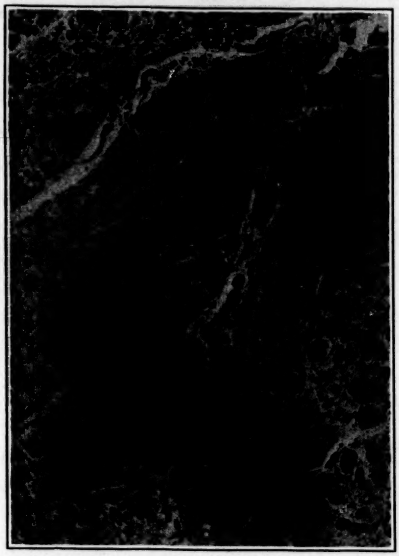

FIGURE 2. *Photomicrograph of Heart Muscle.*

gm., some might consider it to be at the upper limit of normal. The dilatation was equal in both the right and left chambers. The valves were negative. The coronary arteries were smooth, pliable and widely patent, and showed only a few atheromatous plaques. A small mural thrombus was found in the left auricular appendage, but no thrombi were detected in the other chambers of the heart. The heart itself was quite flabby, and the myocardium of both the right and left ventricles was rather gray in color, especially the trabeculae carneae and some of the papillary muscles. The liver was small, weighing only 1100 gm., and showed no evidence of cirrhosis. The spleen was small, weighing 160 gm., and contained two or three healed infarcts; it is quite possible that at one time the spleen was larger. One of the kidneys also had several healed infarcts.

Sections of the heart showed microscopically an unusual condition, which I am at a loss to explain. There was a diffuse interstitial fibrous myocarditis, involving both the right and left chambers but slightly more marked on the right (Figs. 2 and 3). Fibrous connective tissue had replaced a large amount of heart muscle, but its presence around individual bundles and its widespread distribution are quite unlike the scarring due to coronary insufficiency. It is not Fiedler's myocarditis because there is no cellular infiltration. Neither the large vessels nor the small arterioles showed any change. Beriberi or another type of vitamin deficiency seems unlikely. He had had the disease a long time, and I believe that he had had an adequate diet. We made a number of sections of various muscles of the body, all of which were normal. We thought seriously of dermatomyositis involving the myocardium. I suppose all one can call this is interstitial myocarditis, type undetermined.

DR. BURWELL: The patient did not have the circulatory picture of beriberi, low pulse pressure, a low circulation time, and no complications to go with it.

DR. WHITE: Could this be a residual from some such condition as was found in a case I discussed at a recent clinicopathological conference at the Peter Bent Brigham, a case of fatal influenzal myocarditis? Such is doubtless rare when of high degree, but it is possible that we are failing to recognize a few

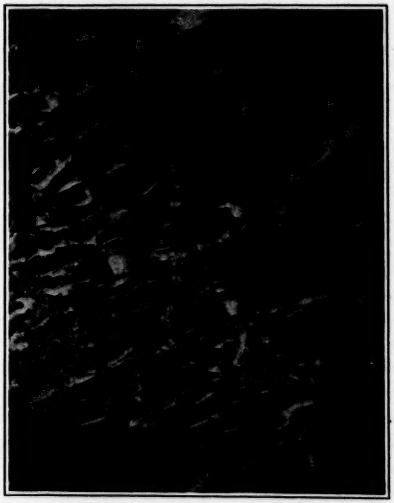

FIGURE 3. *Photomicrograph of Heart Muscle (higher magnification).*

cases of serious myocardial involvement following influenza or other virus diseases.

DR. CASTLEMAN: Any acute infection that he had years ago that involved the myocardium could have produced this picture in the healed stage.

DR. BURWELL: The patient did not have vascular disease. The arteries were fairly good. The absent

pulsations in the feet were not borne out by anything that was found.

DR. CASTLEMAN: The aorta was normal.

DR. WHITE: In a recent article, Levy and von Glahn,[6] of New York City, review a series of cases of myocardial disease of unknown cause. There are certain aspects of this case of ours that are somewhat similar to those of some of their group.

DR. MEANS: Dr. Castleman said that our patient had no hypertrophy.

DR. CASTLEMAN: I doubt that it fits in their group. I should rather believe that it was a deficiency affair or the end result of an acute infection years ago.

DR. MEANS: What about diphtheria? Does that end up with anything of this sort?

DR. CASTLEMAN: I believe that the myocarditis would be more spotty.

DR. BURWELL: What did you find in the brain?

DR. CASTLEMAN: Nothing was observed grossly or microscopically.

DR. WHITE: A word about the electrocardiogram. The low voltage of all the complexes in the limb leads is the most extreme that I have ever seen. The chest leads also indicate widespread myocardial disease.

REFERENCES

1. White, P. D., and Churchill, E. D. Relief of obstruction to circulation in case of chronic constrictive pericarditis (concretio cordis). *New Eng. J. Med.* **202**:165-168, 1930.
2. White, P. D. Chronic constrictive pericarditis (Pick's disease) treated by pericardial resection. *Lancet* **2**:539 and 597, 1935.
3. Moritz, F., and von Tabora, D. Über eine Methode, beim Menschen den Druck in oberflächlichen Venen exakt zu bestimmen. *Deutsches Arch. f. klin. Med.* **98**:475-505, 1910.
4. Chapman, E. M., Dill, D. B., and Graybiel, A. Decrease in functional capacity of lungs and heart resulting from deformities of chest: pulmonocardiac failure. *Medicine* **18**:167-202, 1939.
5. Harrison, M. B., and White, P. D. Chronic constrictive pericarditis: follow-up study of 37 cases. *Ann. Int. Med.* **17**:790-806, 1942.
6. Levy, R. L., and von Glahn, W. C. Cardiac hypertrophy of unknown cause: study of clinical and pathologic features in 10 adults. *Am. Heart J.* **28**:714-741, 1944.

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THE STRAYER SURVEY AS IT CONCERNS SCHOOL HEALTH IN BOSTON

The recently published Strayer survey of the Boston public schools is having its repercussions, and it is to be hoped that some definite improvements may be accomplished before the echoes die away. Surveys in themselves are excellent, but they are chiefly fact-finding and oblivion is their usual fate unless some agency or agencies take on the responsibility of following through with the recommendations.

Physicians should be particularly interested in the section of the report that deals with health services and health and physical education, and its criticism of health education has already received editorial comment in the *Journal of the American Medical Association** — it is not that this phase of Boston's school activities is good, so much as it is that other cities may be no better.

Three main faults, in particular, are to be found with health instruction in the Boston schools, but these three are fundamental. According to the report, "The teachers are not trained in this subject, or only partly trained, or in some cases badly trained; textbooks and other vital teaching aids are lacking; and there is no adequate curriculum." Health service, as another factor in health education, although furnished by competent physicians and a staff of excellent nurses, with an advisory committee of high caliber, suffers from overdetailed legal prescriptions and inferior physical equipment. All health-service activity is authorized by law: the school physicians, for example, are required separately and carefully to examine every child in their respective districts at least once in every school year and to send a written notice of any defect or disability requiring treatment to the parent or guardian. The result is a burden of rapidly performed and superficial annual physical examinations, whereas careful examinations performed once in three years would be of incalculably greater value. The present system may be considered to be antiquated and inadequate.

The functions and purposes of physical education are largely misunderstood. Thus, in a school document presenting a course in physical education it is stated that the purpose is to "provide an invigorating change from seat work." Four basic functions of physical education are given: to ensure the wholesome development of the vital organs of children; to teach functional skills; to develop and maintain an interest in various forms of motor activity; and to assist in the development of standard ways of behaving. Physical educators enjoy the reputation of having an aggressive attitude toward life, and in various instances have assumed or have been given the responsibility for health instruction, for which they are not trained, in addition to that for physical education.

Trained personnel, nevertheless, stands out as the greatest need in health instruction. The duty

*Editorial. Health education in Boston schools. *J. A. M. A.* 126:1086, 1944.

usually falls to the lot of the room teacher. If she is not trained for this part of her job, it is up to the teachers' colleges of this health-minded nation to see that teachers of the future are so trained, just as it should be the responsibility of the medical schools to see that medical students are taught to understand and appreciate the duty that medicine owes to the school child.

In Massachusetts, well-organized and well-staffed state departments of Health and of Education have been engaged in the mutual enterprise of offering constructive health-education programs for the cities, towns and villages outside of Boston that care to participate. A joint committee in 1941 and 1942 made a study of health instruction in the secondary schools, and a relatively permanent joint steering committee has prepared courses of study for the various grades. The departments function largely in an advisory capacity and have, unfortunately, had difficulty in obtaining sufficient funds from the Commission on Administration and Finance to carry on these activities. The revised *Guide to School Health*, for instance, went to the commission on August 23, 1944, but as yet no action has been taken.

Boston, although in some ways a part of the Commonwealth, does not recognize its good offices, being a law unto itself. If Boston is considered the right hand of the Commonwealth, and the state outside Boston the left hand, then the right hand knoweth not what the left hand doeth, although the left hand doeth very well in many matters. Were it not too much *infra dignitatem*, the capital city might benefit from a scrutiny of these departments' activities.

THE MEDICAL CLASSIFICATION SCHEME OF THE BOSTON MEDICAL LIBRARY

Soon after the Boston Medical Library was organized on August 20, 1875, with Dr. Oliver Wendell Holmes as president and Dr. James R. Chadwick as librarian, two rooms on the first floor of No. 5 Hamilton Place were fitted up as a library, under the charge of Dr. Edwin H. Brigham as assistant librarian. A scheme of classification of books and pamphlets was quickly adopted, only to be remodeled four years later by Dr. Chadwick, ably assisted by Mrs. Elizabeth J. Collins, the cataloguer. The new model was based, with important modifications, on the *Index Medicus*, a monthly classified record of the current medical literature of the world compiled under the supervision of Drs. J. S. Billings and Robert Fletcher at the Army Medical Library in Washington. Indeed, at the same time that Dr. Billings was at work, with Dr. Fletcher, on the first volume of the *Index-Catalogue of the Library of The Surgeon-General's Office, United States Army*, Dr. Chadwick, in Boston, was reading proof for Dr. Billings of that vast compilation. Thus, medical classification was much in the minds of these ardent gentlemen and the Boston Medical Library was well in the forefront of a rapidly evolving subject.

As years went on, further changes were needed in the Boston scheme and this work was undertaken by James F. Ballard, then and now the director of the Boston Medical Library. The value of the Boston classification was soon recognized, particularly after it had been published in the January, 1918, issue of the *Bulletin of the Medical Library Association*.[1] The plan was soon adopted as the official classification scheme of the Medical Library Association and was recommended for use in medical libraries, particularly new ones.

The Boston Medical Library classification is now used in all the medical schools and hospitals of Boston and in many medical libraries throughout the world. Over seven hundred copies of a second edition,[2] published in 1925, were sold, thus testifying to the usefulness of this classified plan. A new revision has recently been issued, with a few additions.[3] The scheme is remarkably simple and easy to expand, and has become the most popular for medical-library use. It has been adopted by the libraries of the Cleveland Medical Association, the Mayo Clinic, the Peking Union Medical College and the Colorado University School of Medicine and many others.

The chief disadvantage of the Boston Medical Library scheme has been its infrequent revision, with lack of sufficient expansion in certain sections. These criticisms have been met in the 1944 edition, and in addition to the usual schedules, Mr. Ballard

is preparing an index, thus adding greatly to its value.

References

1. Ballard, J. F. Classification scheme of Boston Medical Library. *Bull. Med. Lib. Assoc.* 7:33-63, 1918.
2. *Idem. Boston Medical Library Medical Classification.* Second edition, revised and enlarged. 44 pp. Boston (privately printed), 1925.
3. *Idem. Boston Medical Library Medical Classification.* Part I: Schedules. Third edition, revised. 46 pp. Boston (privately printed), 1944.

MISCELLANY

DR. POLLOCK RETIRES

Dr. Henry M. Pollock retired on February 1, after thirty years as superintendent of the Massachusetts Memorial Hospitals. Dr. Pollock had been a hospital administrator for forty-six years, having come to the Massachusetts Memorial Hospitals after eleven years of successful administration at the Norwich State Hospital. The enviable reputation made in Norwich continued during his superintendency at the Massachusetts Memorial Hospitals. Besides many innovations and improvements in all departments, he was largely responsible for the addition of two new buildings to the plant. He was a constant crusader for the elevation and maintenance of medical standards, education and practice. His activities in the hospital field include many years as associate commissioner of the Department of Mental Diseases (now the Department of Mental Health); he was a trustee of the American Hospital Association for six years and is nationally known as a consultant in hospital planning and construction.

Dr. Pollock is succeeded by Dr. Leverett S. Woodworth, formerly associate director of the Harper Hospital in Detroit, president of the Michigan Hospital Association and a trustee and secretary of the Greater Detroit Hospital Council.

CORRESPONDENCE

DELETERIOUS EFFECTS OF PRIVINE HYDROCHLORIDE

To the Editor: Privine hydrochloride, as 0.10 and 0.05 per cent solutions, was introduced in 1942 as a nasal vasoconstrictor. The advantages claimed for Privine were prolonged vasoconstriction, small dosage and physiologic rationale. The dose of Privine is two to three drops nasally, and it produces symptomatic relief from the nasal congestion for two to six hours. Since it is prepared in isotonic solution buffered to the same pH as that of the "delicate nasal mucous membranes," it was said to be nonirritating; furthermore, it was claimed that it restores the alkaline pathologic secretions to the normal acid range.

Almost overnight Privine became the popular nose drop. During the last year I sensed the fact that more and more patients were becoming chronic users of Privine and began to see a number of patients who had either marked vasomotor reactions superimposed on the coryza of hay fever or definite exacerbations of symptoms in cases of typical hyperplastic vasomotor rhinitis. These patients generally gave a similar story. Privine was prescribed either for a "head cold" or for some form of allergic coryza. At the beginning they found that one to two drops of Privine instilled nasally gave excellent relief for two to six hours. In fact the majority thought that this was the finest nose drop they had ever used. As the patients continued to use the medication, however, they found that the nasal congestion began to recur as often as every one or two hours. Some soon began to believe that they could not get along without the use of Privine and would take out a vial to show that they kept it with them at all times.

A frequent observation following the use of Privine for hay fever was the continuance of symptoms far beyond the pollinating season. This was most unusual and was commented on by a number of "Privine addicts." In many cases the Privine had not been prescribed by a physician. For example a woman whom I saw recently gave a history of ragweed-pollen hay fever of five seasons' duration. Because of nasal congestion she purchased a bottle of Privine over the counter at her local drugstore on September 16, 1944. This past fall, for the first time, she did not experience the usual relief of hay-fever symptoms with the coming of frost. It is interesting to note that since she first started to use Privine she has been able to purchase ten bottles of this solution.

Nasal examination in practically all these cases reveals turgescence, pallor and bogginess, with much watery secretion. The appearance is almost identical with the picture of the chronic hyperplastic types of vasomotor rhinitis with a superimposed vasomotor reaction.

I have never seen such reactions and such an addiction following the use of ephedrine and the synthetic astringents. Perhaps these lack the dramatic vasoconstricting effects of Privine, but nonetheless patients do not become chronically habituated to their use. The treatment of the vasomotor effects resulting from the continued use of Privine is simple: discontinuance of the drug in nearly all cases gave almost complete relief in seven to ten days.

I believe that Privine should never be used for longer than four to five days and then only when the patient is under the care of a physician. Furthermore, something should be done about the indiscriminate sale of Privine over the drugstore counter.

Irving W. Schiller, M.D.

520 Beacon Street
Boston 15

STANDARDS OF MEDICAL EDUCATION

To the Editor: I have read with interest the editorial "Standards of Medical Education," which appeared in the February 1 issue of the *Journal.* May I comment on it, as it shows some lacunae which the medical profession may wish to have filled, and some errors in statement of fact which, while not of vital import, should be corrected in the interests of accuracy.

The editorial does not do justice to the Approving Authority in saying that "the failure of the Massachusetts Approving Authority to accredit the Middlesex University School of Medicine was upheld by the Superior Court of Suffolk County." The failure on the part of the Approving Authority, if there was any failure, would be in the procedure leading to the actual decision and not in the omission of a decision, as the writer of the editorial implies. A definite decision was made, with full knowledge that the decision meant, as far as the Approving Authority was concerned, that a medical school which had been growing vigorously and improving itself rapidly, with a faculty and students, and with property and equipment estimated at one and a quarter million dollars, book value, would be closed at a time when the national need for more physicians is urgent.

The bearing of the personal references in the editorial is not entirely clear, but in the spring of 1935 the secretary of the Massachusetts Medical Society informed me as secretary of the Board of Registration in Medicine that the president of the Society, a past-president and he himself, having been advised by a high political authority, were opposed to the introduction, for the 1936 session of the Legislature, of a bill to exercise some control over medical education, and he urged that no bill be introduced. The matter was considered by the Board at one of its meetings, and, notwithstanding the objection by the officers of the Society, the Board introduced a bill which, however, cannot be recognized in the Act of 1936.

Reference is made in the editorial to Middlesex University and "three inspections." It is worthy of note that after the first inspection, in 1941, there were six points, out of a possible seventeen, against the school; after the second, in 1943, there were five; after the third, in 1944, there were four; and after Judge Cabot's decision there were three. If in three years the school removed 50 per cent of its deficiencies, why was it "necessary for the present Approving Authority to deny approval"? This is a pertinent question for which a public answer by the Approving Authority would not be out of place. The chairman of the Approving Authority is reported to have said that he did everything that he could for the school, that the Approving Authority had no discretion and that it had to act in accordance with the law. Accepting the first statement at its face value, and the third statement as

obvious, there remains to be discussed, first, what the law does or does not say, and second, what the law does or does not mean. I can only touch on some of these points.

At least three defects of the law are evident at once. As Judge Cabot said, "The statute lays down neither for the Approving Authority nor this court any guide by which approval should be granted or withheld."

A second defect is that if approval is once given, it cannot be taken away. No matter how much the school may deteriorate, the Approving Authority can only sit by and let it go downhill, with approval. This would put the Approving Authority in a ridiculous position.

A third defect is that if a school meets the requirements 90 per cent, the law provides for no status intermediate between approved and not approved. Such status does in fact exist, namely, "on probation," although it is not recognized by the statute. In the requirements for approval drawn up by the Approving Authority in 1936, an attempted makeshift for the solution of the problem caused by this defect was introduced by using the word "adequate" in eight of the seventeen requirements. A school may be adequate in giving a good medical education, although inadequate in one or more of its departments. Few schools satisfy even themselves in every respect.

When the question was raised as to whether the makeshift solution would be satisfactory, a bill was introduced before the Legislature to cover this very point but it was not enacted. Yet right here is perhaps the most serious defect in a very defective law.

The writer of the editorial is too optimistic in saying "At long last the practice of medicine in the Commonwealth is open only to the men and women who have been properly trained and Massachusetts need no longer be called 'the dumping ground for medical-school graduates'." Apparently a clerical error has slipped in here as since about 1914, only medical-school graduates have been admitted to examination. But the writer has forgotten that every physician who matriculated in a medical school before January 1, 1941, no matter how long before, is eligible for admission to examination, even if the medical school from which he graduated has never been approved.

On the other hand, the writer of the editorial is too pessimistic. Referring to bills now before the Legislature, he says, "The passage of such legislation can either halt or stop forever the progress toward higher medical standards." "Forever" is a long time. Does he think that the General Court of Massachusetts can, in 1945, take any action that will last forever? Then he goes on to say, "and high standards of medical education *must* be maintained in these days when the practice of medicine is being subjected to criticism." *Must* they be maintained? Let him see the Army about that and then talk with some of the persons in medical education who are vocal on this subject, such as Dean Branch, Dr. Evarts Graham, Dr. Victor Johnson, Dean MacEwen, Dean Rappleye and Dr. Joseph Wearn, and see if what *must be* done *has been* done.

The many defects of the Act of 1936, Chapter 247, I shall take up at another time, for they are serious and should be remedied. Some of the bills which have been introduced incorporate suggestions for improvement. There is needed a discriminating understanding of what the situation demands. We are in the transition period of a reform, and every reform presents the possibility of at least two dangers — first, from fanatical reformers who ruthlessly seek the desired end without regard for the harm they may do on the way, and second, from the self-seeking who may profit from the reform and to whom justice is meaningless as applied to persons other than themselves. Against both dangers we should be on guard now.

STEPHEN RUSHMORE
520 Commonwealth Avenue
Boston

* * *

The *Journal* is primarily interested in the improvement of standards of medical education in Massachusetts, rather than whether or not this or that medical school should be accredited. Few will deny that in 1936, by virtue of its inadequate medical-practice act, the Commonwealth had become a "dumping ground for the graduates" of non-approved medical schools. Certainly those who are responsible for the health of the people should undergo a course of training that meets certain minimum standards, and the creation of the Approving Authority was a step in the proper direction. It is to be hoped that future legislation will continue forward rather than turn backward. — ED.

NOTICES

NORTON MEDICAL AWARD FOR 1946

The Norton Medical Award, which is offered annually by W. W. Norton and Company, Incorporated, to encourage the writing of books on medicine and the medical profession for the layman, totals $3500 ($1000 outright and the balance an advance against royalties). The author must be a professional worker in the field of medicine, or a collaborator with such a person. The final date for submission of manuscripts is December 1, 1945. Further details are contained in a descriptive circular that may be obtained from W. W. Norton and Company, Incorporated, 70 Fifth Avenue, New York 11, New York.

BOSTON SOCIETY OF BIOLOGISTS

A meeting of the Boston Society of Biologists will be held in the Main Lecture Room of the Harvard Biological Laboratories on Wednesday, March 21, at 8 p.m.

PROGRAM

Estrogen Metabolism in Women. Drs. O. W. Smith and G. Van S. Smith.

Vitamins in Relation to Skeleton Growth. Dr. S. B. Wolbach.

Chorionic Luteotropin and Luteal Function in Monkeys. Dr. F. S. Hisaw.

GREATER BOSTON MEDICAL SOCIETY

A meeting of the Greater Boston Medical Society will be held in the auditorium of the Beth Israel Hospital on Wednesday, March 21, at 8:15 p.m. Dr. Philip Levine will speak on the subject "The Clinical Significance of the Rh Factor." A discussion by Drs. Benjamin Alexander, William Dameshek and Karl Singer will follow.

NEW YORK INSTITUTE OF CLINICAL ORAL PATHOLOGY

The New York Institute of Clinical Oral Pathology will hold an open meeting at the New York Academy of Medicine, 2 East 103rd Street, New York City, on Monday, April 30, at 8:15 p.m. A symposium entitled "A Survey of the Antibiotic Problem" will be presented. The program is as follows:

The Role of Biotics and Antibiotics in Chemotherapy. Dr. Daniel Laszlo.

The Problem of Penicillin in Treatment of Mixed Infections. Dr. Frank Lamont Meleney.

The Treatment of Acute and Chronic Infections of the Jaws with Antibiotics. Dr. Leo Stern.

The Relative Value of Antibiotics in the Treatment of Otolaryngic Diseases. Lt. Col. Kenneth M. Kahn.

Evaluation of Antibiotic Agents for Root Canal Treatment. Dr. Louis I. Grossman.

The Role of Iontophoresis in Ocular Therapy with Antibiotics. Dr. Ludwig von Sallmann.

Treatment of Ulcerative Stomatitis (Vincent's Infection) with Penicillin. Dr. Alvin E. Strock.

For further information all communications should be addressed to G. Roistacher, Executive Secretary, 101 East 79th Street, New York 21, New York.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, MARCH 22

FRIDAY, MARCH 23

*9:00–10:00 a.m. Some Aspects of Cardiac Surgery. Dr. Robert E. Gross. Joseph H. Pratt Diagnostic Hospital.

*9:00–10:00 a.m. Medical clinic. Isolation Amphitheater, Children's Hospital.

10:50 a.m. Postgraduate clinic in dermatology and syphilology. Amphitheater, Mallory Building, Boston City Hospital.

(*Notices continued on page xvii*)

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Indicated in the symptomatic and prophylactic relief of asthma and other allergic conditions*

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*A New Type of Medication to Be Used in Bronchial Asthma and Other Allergic Conditions. *New England Journal of Medicine* 223:843-846, 1940.

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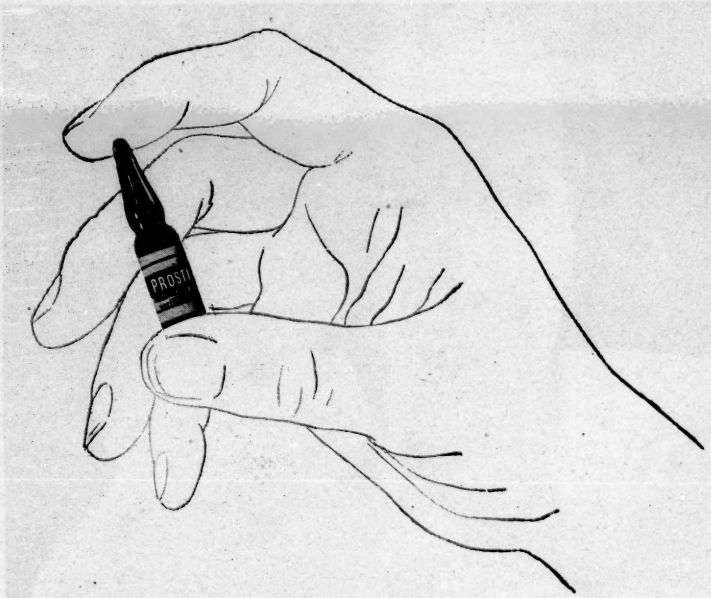

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